OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

VIRGINIA MEDICAL MONTHLY

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The State Board of Medical Examiners of Virginia

Will hold its next meeting at the Richmond Hotel in Richmond, December 5, 1951. The examinations will be held in the same hotel December 6, 7 and 8. All applications and other documents pertaining to the examination, or to matters to be discussed by the Board, must be on file in the secretary's office on or before November 24, 1951. The secretary of the Board is Dr. K. D. Graves, 631 First Street, S.W., Roanoke, Virginia.

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Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance. [1,2] Such states may involve any one of the organ systems or several at one time. [1,3] The outline below is designed to relate gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	Parasympathetic
Gastro-intestinal Sytem	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio-vascular System	Rapid heart rate Peripheral vaso-constriction	Slow heart rate Vasodilatation
Functional Manifesta-tions	Palpitation Tachycardia Elevated blood pressure Dry mouth and throat	Heartburn Nausea-vomiting Low blood pressure Colonic spasm

The data here tabulated is from references 3,4,5,6,7, given below.

When the clinical picture is suggestive of functional disorder, the diagnosis is supported by the presence of the following indications of autonomic lability:

Variable Blood Pressure
Body Temperature Variations
Changing pulse rate
Deviations in B. M. R.
Exaggerated Cold Pressure Reflex
Oculo-Cardiac Reflex Abnormalities
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relieving the somatic disturbance to prepare the patient for psychotherapy*; 2) guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

*Drug treatment using adrenergic and cholinergic blocking agents in conjunction with sedatives. 8,9,10.

1. Ebaugh, F.: Postgrad. Med. *4:* 208, 1948. 2. Wilbur, D.: J.A.M.A. *141:* 1199, 1949. 3. Williams, E. and Carmichael, C.: J. Nat'l. Med. Assoc. *42:* 32, 1950. 4. Goodman, L. and Gilman, A.: The Pharmacological Basis of Therapeutics, The Macmillan Co., 1941. 5. Katz, L. et al: Ann. Int. Med. *27:* 261, 1947. 6. Weiss, E. et al: Am. J. Psychiat. *107:* 264, 1950. 7. Alvarez, W.: Chicago Med. Soc. Bulletin, 581, 1950. 8. Rakoff, A.: A Course in Practical Therapeutics, Williams and Wilkins, 1948. 9. Karnosh, L. and Zucker, E.: A Handbook of Psychiatry. C. V. Mosby Co., 1945. 10. Harris, L.: Canad. M.A.J. *58:* 251, 1948.

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GUEST EDITORIAL

The Need for "Heart Specialists"

ANGIOCARDIOGRAPHY, cardiac catheterization and multiple leads in electrocardiography with their interpretation by analytical geometry have combined to fill with humility the soul of the physician who was graduated before World War II. His humility may at times become so deep that he can almost agree with the post-war graduate that he knows nothing at all about the heart.

How much of this inferiority complex is justified? What is necessary to make a reliable diagnosis of heart disease?

In the first place, in most cases the history is the most important single factor in making the diagnosis. The examiner must always take a careful history. Paul White has said that if he had to choose between an assistant taking the history and himself examining the patient, and White taking the history with the assistant making the physical, White would take the history. He greatly preferred, if he were to accept the responsibility for the patient, to do both.

To take a good history one must be first of all a *doctor*. Nothing is more to be deplored than to hear a third year student announce that he is going to be a "heart specialist" unless it is to see him allowed to continue his training with no exposure to other subjects.

Is the "shortness of breath" the woman complains of merely the sighing type of respiration characteristic of psychoneurosis, especially in fat middle-aged women with tight girdles? A stomach full of air may produce discomfort that the kind of patient who swallows air excessively will attribute to her heart. Is it hysterical hyperventilation? Does he wheeze? Does he wheeze all the time or only when he tries to sleep with a single pillow?

Or is it the real dyspnea of effort? How much effort is necessary to provoke it? Is less effort required now than six months or a year ago? How much physical activity does he engage in? Many people, especially among those over 40, don't enjoy exercise! Would he do more if he could?

How does he sleep? Can he sleep flat? Has he used two pillows for years? Does he have night starts? Swineford has distinguished two kinds of "cardiac asthma": in one the patient wakes with a start, his chest full of moisture and not a wheeze to be heard; in the other, wheezes obscure moisture, but no wheezes will be heard in the daytime. Both need dehydration, but the second also needs a plastic pillow case. Some persons wake up at night for no better reason than that they went to bed worrying. Does he, between the time he turns off the light and the time he falls asleep, feel his heart "stop for a second", or perhaps "turn over" or suddenly give a single beat "that shakes the whole bed"? Does his heart sometimes abruptly begin to race and quiet down with equal suddenness or does it slow down imperceptibly?

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Has he ever noticed any swelling around the ankles? When? Was there any swelling around the eyes? Does his occupation keep him on his feet relatively motionless all day? Is the swelling only present in hot weather? When was the last time his urine was checked?

Does he have chest *pain*? Where is it? Pain over the apex is almost always *not* associated with the heart. Does it radiate? What brings it on? Has he ever suffered agonizing chest pain for hours, accompanied with drenching night sweats and, often, restlessness?

Has he ever had rheumatic fever? It is well to use synonyms, and to ask about nosebleeds, repeated sore throats, and other relevant symptoms. Has he ever had syphilis? Is there a family tendency to high blood pressure, strokes, or sudden death? Has his own blood pressure ever been found high? When was it last taken?

How is his digestion? Does he ever vomit? Has he ever passed tarry stools? Suffered with bleeding piles?

Has he lost weight? Is he very emotional? Has he always been a worrier or did he just a few months ago for the first time in his life become very nervous, with tremor and excessive appetite, weakness and diarrhea? Does she menstruate excessively?

These questions are, of course, not all that should be asked. In the individual case, the doctor may find clues to lead him on to many others. But by the time the history is completed, if the doctor has not arrived at a tentative diagnosis he should at least know what sort of a person he is dealing with.

The man who begins the examination of a patient by slapping his stethoscope over the apex of the heart betrays he knows little about the heart. The first thing he should do is to look his patient over. Is he pale? If his hemoglobin is less than 8 gm., the least exertion may exhaust him, but he can probably sleep without a pillow. Or is he plethoric? Is cyanosis or clubbing present? Is he very fat? Has he lost weight rapidly? Does he have a stare or a tremor? Are varicose veins responsible for swelling of his legs? Or tight garters? Do large veins converge on the navel? Is there fluid in his belly? Is the liver enlarged? Is there tenderness along the right costal margin? Is there a mass arising from the pelvis to be felt in the lower abdomen? Is splenomegaly or lymphadenopathy present?

In the conjunctivae or retinae it may be possible to find the tiny hemorrhagic emboli characteristic of subacute bacterial endocarditis. Lemon sclerae may evidence pernicious anemia or the sclerae may be bile-stained. Funduscopic examination is of greater value in diagnosing the severity of hypertension than a single blood pressure determination. Are the pupils normal?

Is there a precordial thrill or abnormal pulsation over any of the arteries? Is the heart enlarged? Fluoroscopic examination is of great help. When the patient is under thirty, a heart normal in size and contour, it is pretty safe to say is a normal, healthy heart; in a middle-aged person however, the chances are only two to one that a heart that looks all right is as good as it looks.

Books have been written about auscultation. Suffice it here that it is often possible to diagnose the type of irregularity by this method, as well as to determine accurately the presence of valvular disease. An electrocardiogram will not determine the valve involved; indeed it is of little use in chronic valvular disease. It must be added that, in school children at least, only about one murmur out of ten is important, and not

more than one of the others is worth periodic check-ups. The presence of a murmur then is *not* an indication for digitalis.

While the importance of taking the blood pressure has been exaggerated, it should always be done.

An iatrogenic cardiac neurosis is very hard to treat. And most of all cardiac neuroses are caused by a doctor who may have appeared to the patient uncertain as to the diagnosis or seriously concerned over his condition; or the patient may have misunderstood something he said. It is also possible the doctor may have been mistaken: the error of attaching importance to a normal variation from the usual electrocardiogram is much commoner than the error of overlooking something of significance. The most difficult type of case is one in which there is actual heart disease complicated by severe neurosis. Such cases are not uncommon. And often the neurosis causes more trouble than the actual cardiac lesion.

There is at least one doctor in the United States who still delivers cataracts and babies with equal aplomb. But it is likely that a man whose interests are not so wide will ruin fewer eyes and fewer perineums. By that same token, one who limits his activities to internal medicine may handle certain cases more efficiently than one whose horizon is unlimited.

Examination of the blood and urine should always be done, and such examinations can be done by any physician. An x-ray of the chest or, perhaps better, a fluoroscopic study is desirable.

The question of an electrocardiogram arises in almost every case of heart disease, real or otherwise. If the patient is greatly worried over his heart, though he is perfectly sound infratentorially, a tracing is always indicated but if the easy exhaustion is the result of acute leukemia or a bleeding fibroid it is hardly necessary. There is a great deal to be said however, for routine electrocardiograms in persons over 45. Tracings are useful in the management of active rheumatic fever and coronary disease. In these conditions serial tracings are vastly more important than single ones. The first one taken on a rheumatic young person may appear within the limits of normal until compared with one taken several months after he has recovered. Similarly a cardiogram on a man of 60 which appears alarming may be unchanged after ten more years of productive life.

In this day of successful correction of several cardiac anomalies, and the dawning of the day of successful operations on the valves themselves, one owes it to a child handicapped with congenital heart disease, to send him to a medical center where specialists can do every type of examination possible to determine whether or not he is a good candidate for operation. This does not mean that every child with a heart murmur need be so referred.

L. MINOR BLACKFORD, M.D.

Dr. Blackford is a graduate of the University of Virginia, Department of Medicine, and is practicing internal medicine in Atlanta, Georgia.

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A BRIEF HISTORY OF THE MEDICAL SOCIETY OF VIRGINIA AND ITS FUNCTIONS*

C. LYDON HARRELL, M.D.,
Norfolk, Virginia.

Members of The Medical Society of Virginia, Invited Guests, Ladies and Gentlemen:

I wish to thank you for the honor and privilege of being permitted to serve you as President during the past year. This year has been a hard and trying one, but it has brought pleasures and experiences that I will never forget. It has not been a full time job, but it certainly has been an everyday job. One of the duties of the retiring President is to deliver an address on the opening night. I have chosen as my subject, "A Brief History of the Society and its Present Functions."

The object of any medical society is to bring its members together for a better understanding of each other, to promote public health, to collect and disseminate medical knowledge, and to help each other in every way to better serve his community and mankind. Every physician, as soon as he locates in practice, should join his local and state Medical Society and the American Medical Association. He will not only help himself but also will help those that are already members to do a better job.

On December 15, 1820, The Medical Society of Virginia was first organized by a group of seventeen physicians from Richmond and Manchester, now "South Richmond". Dr. James McClurg, of Richmond, was elected as the first president. The first charter of The Medical Society of Virginia was passed by the General Assembly of Virginia, January 2, 1824. This charter permitted the Society to charge admission fees and annual dues and sue for same, if not paid. So far as I know, this privilege has never been exercised. It also permitted the Society to buy and own real property for its personal use. It also gave authority to sublet a portion of the property not in use, provided the net income does not exceed two thousand dollars per annum over a period of 10 years.

The Society met regularly until November 1824, at which time it passed into a state of coma; cause unknown. According to Dr. Blanton, on December 27, 1841, seventeen years later, the Society was revived, the old constitution readopted, and new officers elected. It continued to function until 1859 when the Society had another period of lethargy, probably due to a resolution advocating a board of medical examiners. (For a more detailed description see Blanton's "Medicine in Virginia in the 19th Century".) However, on November 2, 1870, the local societies of Lynchburg, Richmond, and Abingdon held a convention and reorganized The Medical Society of Virginia and on January 14, 1871, another charter of incorporation was granted. The only difference between this charter and the first charter is that the word "The" is a part of the title and the letter "T" should be a capital. We have been working under this charter since 1871, though December 15, 1820, is the date of the official seal.

Certain members of this Society have always stood for high standards in scholarship and training. As early as 1851 Dr. Goodridge A. Wilson, of Richmond, advocated before The Medical Society of Virginia the creation of a board of medical examiners. This was a frequent and heated question of debate for many years with frequent requests to the State Legislature to pass a Medical Practice Act. The first bill was passed in 1884. That following November 15 the first Virginia State Board of Medical Examiners was organized in Richmond. The first examination was held April 8, 1885. From this time on, it has been the purpose of The Medical Society of Virginia to maintain a high standard of education and requirements of its membership and to approve only those who are graduated from grade A schools.

If I had to mention any one man who had been more influential and who had given more service in making the Society a success, I would name Dr. Landon B. Edwards, of Lynchburg, later of Richmond. Dr. Edwards was elected secretary of the Society in 1871 and served forty-two years. He was succeeded by his daughter, Miss Agnes V. Edwards, who was secretary for twenty-six years. Miss Ed-

*Address of the President before The Medical Society of Virginia at its annual meeting at Virginia Beach, October 7-10, 1951.

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wards has been with the Society thirty-two years. At present she is acting as Managing Editor of the VIRGINIA MEDICAL MONTHLY. The membership of the Society owes a debt of gratitude and thanks to these two people for their long terms of service and untiring efforts in helping to make The Medical ical Society of Virginia the great success that it is.

The VIRGINIA MEDICAL MONTHLY was started by Dr. Edwards in 1874, as a private enterprise. It was sold to The Medical Society of Virginia in 1919 and has been published as its official publication since that date.

The Society used as its headquarters rented space in the Richmond Academy of Medicine Building for many years. We were nicely taken care of there, but, after adding the Department of Public Relations, all available space was entirely too small. It became necessary to seek a new home elsewhere. Following the approval of the House of Delegates last year, the building at 1105 West Franklin Street, Richmond, was purchased in December for our permanent headquarters, and the following April we moved in. We now have ample space to work in and two floors available to rent.

COMMITTEES

All important business and functions of any organization are usually performed by committees. The same applies to medical societies. The personnel of these committees are those most interested in that particular branch of work.

The Medical Society of Virginia has ten standing committees, and, this year, twenty-six special committees. The incoming president has the privilege of naming the chairman of all standing committees, dropping one member and adding one. He has the privilege of naming his own special committees.

At the mid-winter meeting of the Council in January 1928, Dr. John W. Preston, then President, discussed the question of Post-graduate work in Virginia to be sponsored and financed by The Medical Society of Virginia. Dr. J. Shelton Horsley, who also had referred to this in a previous address, was invited to attend this meeting and participate in the discussion. After a free discussion by the members of the Council, Dr. Israel Brown, of Norfolk, made the following motion: that post-graduate courses with clinical material be undertaken in Virginia and sponsored by The Medical Society, that the actual expense incurred be borne by the Society, that the plans be worked out by the President of the Society, Dr. Preston, and by Dr. J. C. Flippin, of the University of Virginia, Dr. W. T. Sanger, of the Medical College of Virginia, and Dr. E. G. Williams, State Health Commissioner. Thus, was born our Department of Clinical and Medical Education.

This committee, through the guidance of Mr. George Zehmer of the University of Virginia, has rendered valuable service for over twenty years. The actual teaching was done by the staffs of the two medical schools in the form of lectures and clinics.

This year Dr. W. C. Caudill, chairman of the committee, suggested we try a new approach to post-graduate work. It seems that the doctors in the rural sections have not been attending the post-graduate courses of instruction as they should. He decided to take the clinics to the doctors. He requested The Medical Society of Virginia to appropriate $1000 to support this project, which the Council did at the January meeting for 1951.

Through the efforts of Dr. Kinloch Nelson, four sessions have been arranged to be held in different rural sections of the state, endeavoring to reach the country doctor and those in small towns who do not have the time to attend meetings in the city. The sessions last from 2 p.m. to 9 p.m. with intermission for dinner. The instruction is by lectures and clinics. The speakers are paid $50 per session plus expenses. Those attending the lectures pay $4 to help defray expenses. Four sessions have been held thus far. I understand that they have been well received and well attended. I do not know of any better way to promote public relations than to let the public know and the country doctors know that their State Society is doing all in its power to keep them informed of the new discoveries, new developments and new techniques in the practice of medicine.

There are numerous other ways in which we are trying to keep physicians informed. Weekly clinics and lectures are being held at both medical schools where all are welcome. The Medical College of Virginia gives a two weeks refresher course every summer for Negro physicians. Dr. David Wilson, chairman of the Committee on Mental Hygiene, has held two all day sessions at the University this year for general practitioners where they discussed certain

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emotional problems as observed by the general practitioner. Dr. Wilson is very anxious to extend this program another year.

The Cancer Committee, with Dr. George Cooper, Jr., as Chairman, working in conjunction with the American Cancer Society, is doing excellent work which covers the entire State. There are eleven approved tumor clinics located in strategic sections of the State. It is estimated that they will hold 700 clinic meetings this year and receive 4000 patient visits. These clinics have three main functions: accurate diagnosis, outlining the best type of therapy, and instruction. Any physician, living in a reasonable distance from these clinics, misses a golden opportunity to learn the latest about tumors both benign and malignant, if he fails to attend.

The American College of Physicians, the American College of Chest Physicians, and the Diabetic Society, each holds a half day session once a year. The larger hospitals over the State conduct a teaching program for residents and internes. These sessions are held three to four times a week throughout the year. All members of the staffs and visiting members are welcome.

The Department of Clinical and Medical Education is performing a most valuable and necessary function by making it possible for all physicians in the State to get the necessary post-graduate training without costing too much time or money.

Any physician who does not believe in or who fails to avail himself of these opportunities to keep posted on new developments and new discoveries in medical science may be likened to the farmer who sticks to his ox cart when automobiles, trucks and tractors are humming by daily. They will be lost in the dust and deserve to be relegated to the dump heap.

MEDICAL SERVICE

In 1948, Dr. Walter B. Martin and Dr. John T. T. Hundley, members of the Committee on Medical Economics, introduced a resolution requesting that the name be changed to a Committee on Medical Service. According to the Constitution, this committee was to have supervision of all matters relating to pre-paid hospital and medical care and rural health.

This committee for the past few years has been headed by Dr. John O. Boyd, Jr., of Roanoke. In the annual report submitted for your approval this year, they recommended that eight specific services be included in the work on Medical Service, listed as follows: Pre-payment Health and Hospital Insurance, Rural Health, the Chronically Ill, the Medically Indigent, Federal Health Programs, Child Health, Industrial Health, and Health Councils. If these groups could get together and co-operate, they could go a long way toward improving the general over-all medical care in this State. It would indeed set up a great bulwark against Compulsory Health Insurance or any branch of Socialized Medicine.

We still hear a great deal about the increasing cost of medical care. The public assumes that the rapid rise in the cost of medical care is largely due to an increase in medical fees. This is not true. The increased cost in medical fees adds a very small percentage to the total in the increased costs of medical care. The increased cost of medical care is due mainly to the increased charges in hospitalization and drugs, but the doctors receive the blame. The only way doctors can be held responsible for this added cost is that they are rendering better and more efficient medical care than has ever been rendered by any group of physicians in any nation in the history of the world. To render this type of service costs money. The public demands the very best and the latest improvements in medical science. The public is directly responsible for the increased costs.

Forty years ago one could get a bed in the Norfolk General Hospital for from $3 to $5 a day, according to accommodations. The drugs were free, no laboratory fee, and no anesthetist fee. Today the charge per bed per hospital day ranges from $8.50 to $17. The laboratory fee is $12.50 for the first ten days, plus $5 to $10 for tissue work. The operating room and anesthetic costs range from $30 to $40 per operation according to time. The average drugs costs are $1.52 per patient day. The tremendous increase in drug costs has taken place in the past two years because of the discovery of antibiotics, cortisone, and ACTH.

From the use of sulfonamides and antibiotics, much of the practice of medicine has been revolutionized in the past few years. The acute infectious diseases of childhood are no longer the "bug-bear" they once were. Acute mastoiditis is a thing of the past. The mortality in pneumonia in both children and adults has been reduced from 35% to about 5%. The

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mortality from acute blood stream infections which once was 100% has been greatly reduced.

One way that we can help to combat the increased cost in medical care is to insist on our patients taking out some form of Voluntary Health and Prepaid Medical Care Insurance, whether it is in a private insurance company or in a mutual company plan.

Grievance Committee

A few years ago your Society organized a Grievance Committee, composed of five past presidents. Each local society in the State was requested to organize a similar committee. I feel that every member of our Society should know this and familiarize himself as to its duties. The public must be informed that such committees are in existence. If the Grievance Committee is put to proper use, it will go a long way towards settling all misunderstandings between patient and doctor, whether they relate to fees or improper treatment. All complaints should be filed in writing with the chairman of local committees. Thus far all cases have been settled satisfactorily.

Tuberculosis Committee

For several years the main function of the Tuberculosis Committee was to keep the Society informed as to what the State Health Department was doing towards tuberculosis control and treatment. This year an entirely new committee was appointed, with Dr. George A. Welchons as chairman, and assigned a specific duty. This committee was requested, after getting permission from Dr. Joseph E. Barrett, commissioner of the Department of Mental Hygiene and Dr. L. J. Roper, then State Health Commissioner, to make a survey of all mental institutions and all penal institutions in the State. The Committee was instructed to report its findings with recommendations as how best to detect, treat, and prevent the spread of tuberculosis in these institutions.

We do not have the moral right, nor is it economical to confine an individual and subject him to a chronic disease that may incapacitate him for life.

A few weeks ago your speaker with a member of this Committee visited the jail of one of our largest cities. The keeper of the jail was very cooperative, took us through the building and gave us all the information at his command. The building was clean, the food, which appeared to be wholesome, was plentiful. There were no flies or other insects to be seen, but the cells were small and dark with only artificial light and poor ventilation. We were told that around 13,000 prisoners passed through that jail in the course of a year. They remain anywhere from twenty-four hours to four months, occasionally a year. A physician was in attendance for acute sickness, but no effort was made to detect contagious diseases or tuberculosis. There was no method of treatment or segregation of the cases known to have the disease. There is no doubt of the importance of remedying this in the jails and mental institutions.

In the mental institutions we found that an attempt was made to detect tuberculosis on entrance. Those so infected were put in a ward to themselves. That was all the treatment that they received except food. Some effort should be made to treat the tuberculous condition along with their mental treatment.

There are many other committees with important functions, but time (and space) will not permit their discussion.

Public Relations

About eight years ago we formed a committee on Public Relations, consisting of Dr. J. M. Emmett, as chairman, Dr. H. B. Mulholland and Dr. I. C. Riggin, then State Health Commissioner. This was practically a new field for state medical societies, a field that all will admit has been too long neglected. Large corporations and public utilities have had their personnel directors and public relations officers for years.

The medical profession also has realized that it is important and necessary if we wish to preserve our freedom and identity, we must inform the public and those we serve what we are trying to do and how we propose to do it. In 1946, Mr. Henry S. Johnson, a member of the State Legislature, was selected as our Public Relations director. Before his untimely death in May, he had become one of the leading Public Relations directors of any medical society in the country. As you know, for several years the general public has been very dissatisfied with the medical profession, although not necessarily with their private physicians.

The public needs to be shown that they have the highest standard of medical care anywhere in the world and it is improving. Our system of the practice of medicine is the most scientific, progressive

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and far-reaching of any system that has ever existed in the history of the world. The epidemics of contagious and infectious diseases have been reduced to a minimum. Life expectancy has been raised from 46 years to 68.2 years in the last half century.

The increase in the life expectancy was brought about mainly by improved health standards such as pasteurization of milk, vaccination, sanitation, and so on, which greatly reduced infant mortality. In 1900 we had approximately 3 million people alive over 65 years of age. In 1950 we had about 11 million over 65 years of age. With improved health standards comes added responsibility of increased numbers of the chronically ill and old people. We will meet this problem as we have met others. The high standard of medical practice which we have was produced by organized medicine, the medical profession, the medical schools, and the drug manufacturers working together in practice, teaching and research. The success which our system of medicine has attained could not have been accomplished by any one group alone, but only by all working together.

We frequently hear that there is a shortage of doctors. There is no shortage of doctors. We have more physicians to our population than any other country in the world, except Israel. We have one doctor to about every 750 individuals. Because of the influx of physicians from Europe following the war, Israel has one doctor to every 350 individuals. The problem is that the geographical location of our physicians is unbalanced at present. Some cities have too many doctors, which is to the detriment of the rural areas. This may be attributed mainly to two things: the attractions in the cities and specializing by doctors. Cities have cultural advantages, higher standards of living, conveniences, improved roads and methods of transportation, and congestion of people, all of which make it more appealing to the doctor and his family. He feels that he can make more money in a shorter time. This is a false illusion when one stops to consider the added office expense and increased cost of living. Too many doctors, especially young doctors, are taking up specialties, in comparison to those going into general practice. It would be better for all concerned, for the public and for the profession, if every physician were required to do general practice for at least five years before taking up a specialty. He should get a fuller and better all-round knowledge of the science of the practice of medicine before beginning to specialize.

Because of the rising cost of everything in the past few years, we hear much about our medical schools operating in a deficit. Many will have to close unless financial assistance comes soon from some source. I think that this is a fact that cannot be disputed. It is the duty and obligation of the medical profession to help pilot them out of this trouble. The stand taken by the American Medical Association and by the National Foundation for Medical Education is certainly a step in the right direction and deserves our most hearty support.

We certainly do not want the federal government financing medical education. The very day that our federal government begins to assist medical schools our politicians will begin to take control and medical education will start to decay. When they get control, socialized medicine will have been accomplished. We will no longer be able to practice scientific medicine under the standards as we are now doing. This is what has happened in other socialized and bureaucratic countries that have tried it. Once obtained, there will be no turning back.

Thirty-five years ago the medical institutions of Europe were considered the most up-to-date and finest teaching centers in existence. Physicians and medical students flocked there from the United States and from all corners of the earth for post-graduate study. Today the reverse is true. Under their present system their medical schools and standards of medical practice have gradually declined. Students are now coming to America for advanced study where they find the most progressive, most efficient and highest standards of medical teaching centers that have ever been created.

We can maintain this standard only by preserving our free and individual method of thinking and private enterprise, by keeping the politicians out of medicine, and by putting more and better medicine into politicians.

I always felt that it was best that physicians stay out of active politics, that they would do better by sticking to their profession. However, there are some things physicians can do, and should do, to be a good citizen as well as a good doctor. Every physician should vote and see that each member of his family votes. He should encourage all those with whom he comes in contact to vote. A physician should keep

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informed on the political issues of the day. He should inform those with whom he comes in contact what he believes to be right or wrong and inform them as to what he believes to be to the best interest of his community, state and nation. One will find it much easier to convert a candidate before he is elected than after he arrives in Washington.

Senator Richard M. Nixon, of California, delivered an address before the Conference of Presidents and Officers of State Medical Associations meeting in Atlantic City and discussed "A Legislator Views Medicine". He had this to say: "I believe it is essential that all members of the medical profession recognize that an attempt to socialize any American profession — any American institution— constitutes a threat to all. If we understand this, it seems to me that from a selfish standpoint, the members of the medical profession should become interested in political campaigns and remain interested in political affairs, because by objecting to, avoiding, and defeating programs which would socialize other institutions or professions, they will be taking very effective action in behalf of their own interests". He also states, "In a democracy, the character and the quality of our public servants is directly related to the interest and the intelligence of the electorate."

Unfortunately a large percentage of the intellectual people in this country do not take an active part in political campaigns and do not take the time and trouble to vote. They are too interested and too wrapped up in their own private affairs. On the other hand, those who have personal interests at stake, those who are trying to hold their political jobs, and members of trade unions are always found at the polls on election days. They usually get their man elected. If we, as a professional group, wish to have more political weight, we must change our attitude. I again quote Senator Nixon. "The medical profession is made up of a group which represents one of the highest standards of intelligence in the whole country. For that reason, members of the medical profession, I think, owe a duty not only to themselves and to their profession, but also to the nation at large, to give the nation the benefit of that intelligence in selecting the representation the people will have in the state and national capitals. Because if we can increase the general level of intelligence of the electorate, it means that we are going to increase the character and the quality of those who represent us in Washington." This advice applies to the legal profession and to all educated groups alike, if we wish to preserve our standards, our national institutions and our American way of life.

If we wish to preserve our democracy, we must be tolerant of our brother practitioners, regardless of race, color or creed, tolerant of those among us if they can prove their worth, who are qualified and willing to do the right thing. There is a phrase in the constitution of The Medical Society of Virginia, which states that only members of the *white* race may become members of this Society. This means that no member of the Japanese, Chinese, Philippine, Indian or Negro race, no matter how well qualified, could become a member of this Society. This means that because of his color Noguchi, a Japanese scientist, who gave us our present knowledge of neurosyphilis; Hinton, an American Negro, who developed the well known serological tests for syphilis, known by his name; Dr. Khanolkhar, a noted pathologist of India, who has contributed much to our knowledge of tumors, could not join our Society. Fellow members, we should delete the word "white".

It is not fair. It is not just. It is not democratic. It does not manifest the Christian spirit to limit membership in this Society to white people only. If we wish to preserve our freedom and our American way of life, we must think on these things.

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THE "L.E. CELL" A PHENOMENON OF DIAGNOSTIC VALUE IN ACUTE DISSEMINATED LUPUS ERYTHEMATOSUS

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The Hargraves' "L.E." cell is probably the most important recent advance in our knowledge of disseminated lupus erythematosus. This disease in its acute disseminated form at times baffles the most astute clinician. The manifestations of this disease are so variable that the diagnosis often is not made until autopsy is performed. The "L.E." cell, originally observed in 1946 by Hargraves in marrow preparations of patients with acute disseminated lupus erythematosus, has given impetus to further work using this phenomenon as a diagnostic aid.

The purpose of this report is to discuss briefly the cardinal manifestations of this disease and the progress made in utilizing the hematologic aspects of the illness as an adjunct to the diagnosis. Recently the diagnosis of disseminated lupus erythematosus has been substantiated in seven patients by the finding of "L.E." cells in heparinized concentrated bone marrow smears. We are reporting three of these cases in detail, and the remainder briefly.

A review of this disease is not within the scope of this paper. However, several excellent studies have recently been published [1, 10, 14, 16], and should be consulted by those interested. The etiology is unknown and recovery is rare. There is no specific treatment. The disease in its disseminated form has a higher incidence in the Caucasian race and the female is usually affected. It may occur in the Negro race and in males, although the incidence in either is rare. One of the cases which we are reporting occurred in a Negro male. A report of a similar occurrence has recently been published by Vesey and Nelson[15].

Several features of this disease are noteworthy. Fever without any characteristic pattern, a depressed total leukocyte count, and a migratory arthralgia should always arouse suspicion of this disease. Absence of the skin rash, which is supposed to be so characteristic, is known as "lupus sine lupus" i.e., lupus without the characteristic skin lesions.

The "butterfly" skin lesion in this disease is so called because of its distribution over the bridge of the nose and the malar prominences. Exposure to sunlight is considered a *sine qua non* for the development of this manifestation. As the disease progresses, the dematologic lesions become more profuse and may cover the entire body. Ulceration of the fingertips has been stressed as another feature.

Other observations sometimes encountered include polyserositis, various pulmonary findings, gastro-intestinal disturbances, and even central nervous system and cardiac involvement. Atypical non-bacterial verrucous endocarditis (Libman-Sack's disease) is now generally held to be a feature of disseminated lupus erythematosus rather than a separate entity.

Routine laboratory findings usually present in addition to leukopenia include anemia, an increase of the sedimentation rate, a reversal of the albumin-globulin ratio, and false positive reactions in Wasserman or flocculation tests. The kidneys are usually involved and albuminuria and microscopic hematuria are common findings.

In 1946, Hargraves observed a phenomenon in bone marrow preparations of patients suffering from acute disseminated lupus erythematosus which was believed to be of a lytic-phagocytic character. Morton,[11] working under Hargraves' direction, originally reported this in a thesis, and later Hargraves, Richmond, and Morton[2] presented a preliminary report of this finding.

The "L.E." cell acquired its name because of its frequent occurrence in the bone marrow in cases of acute disseminated lupus erythematosus. Characteristically, it is a mature neutrophilic polymorphonuclear leukocyte which has engulfed a round homogeneous, purple-staining mass of chromatic material.

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The cause for such is believed to be either phagocytosis of free nuclear material with a resulting round vacuole containing this partially digested and lysed nuclear material, or, second, an actual autolysis of one or more lobes of the nucleus of the involved cell. Gradations in the process of digestion range from a smoky appearance of "digested" material to relatively intact nuclei where the chromatin pattern is still visible (Fig. 1 and 2).

In seven of the eleven cases of acute disseminated lupus erythematosus reported by Morton, the "L.E." cell was found. In Hargraves' preliminary report, more than twenty-five cases presented this phenomenon. Haserick and Sundberg[7] duplicated this work by finding the Hargraves' "L.E." cell in four out of five cases of acute disseminated lupus erythematosus, and emphasized its diagnostic value especially in those cases in which the classic dermatologic manifestations were lacking. Also, of importance, was the fact that in the subacute and chronic forms of the disease this phenomenon could not be shown. During partial remissions of the acute stage only a rare "L.E." cell may be found where only a short time before large numbers could be found.

Difficulty was encountered by many investigators who tried to duplicate this work. In all cases it was found that the bone marrow smear was a direct smear, whereas Hargraves, Haserick and Sundberg had made their smears according to Schleicher's technic[12] from concentrated and unconcentrated marrow to which an anticoagulant such as heparin, oxylate, or citrate had been added. The question of whether an agent common to all anticoagulants used contained a responsible factor which led to the production of "L.E." cells necessarily had to be settled.

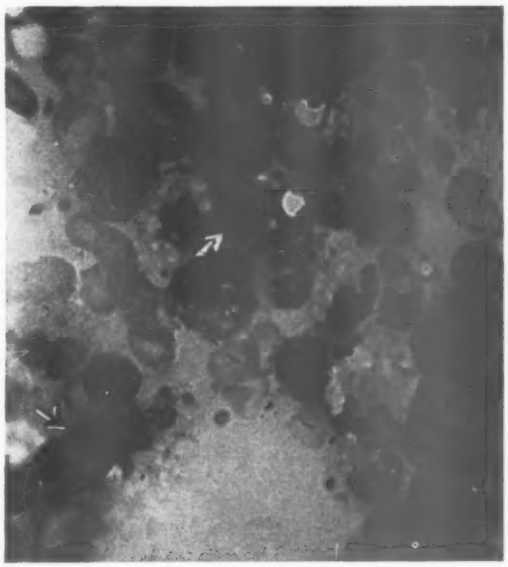

Fig. 1. Concentrated, heparinized marrow from Case 1 (x675). Arrows point to two cells typical of the "L.E. phenomenon".

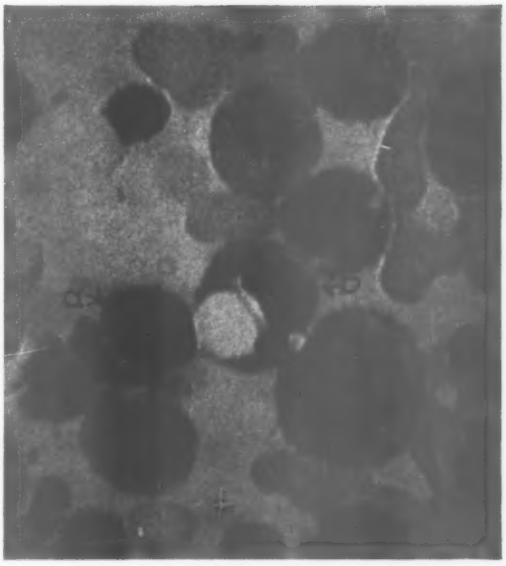

Fig. 2. Concentrated heparinized marrow (x900) from Case 1. (a) Typical cell of "L.E. phenomenon" showing large homogenous mass with remaining nuclear remnants below on periphery. (b) "L.E. cell" in which division of homogenous inclusion body apparently occurred. Note vacuole where previously there was nuclear inclusion material.

This question was brought nearer solution when Sundberg and Lick[13] reported finding of "L.E." cells in the smears of the concentrated leukocytic layer of venous blood in three cases of acute disseminated lupus erythematosus. Moffatt and coworkers have observed typical "L.E." cells and "rosette" formation of leukocytes in the smear of the buffy coat from the mixture of patient's and normal blood. Hargraves[3] showed that concentrated preparations from venous blood showed nucleolysis and agglutination in which neutrophilic leukocytes cluster about the homogenous mass of nuclear material in an attempt to engulf it. This occurrence suggested that rather than the anticoagulant being the factor in causing this phenomenon, it was the result of hypersensitivity. If this hypothesis was true, the agent or factor causing the nucleolysis, agglutination, and

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phagocytosis could be in the plasma of the patients with acute disseminated lupus erythematosus. Hargraves showed this to be true by producing typical "L.E." cells as well as the phenomenon of nucleolysis and agglutination by incubating plasma obtained from the venous blood of patients with acute disseminated lupus erythematosus with bone marrow from patients who did not have this disease. This finding seems to point to the "L.E." cell phenomena being probably immunologic in nature. Haserick and Bortz,[5] utilizing only the plasma of 14 patients acutely ill with acute disseminated lupus erythematosus and heparinized bone marrow of 48 normal persons, induced the "L.E." cell and clumping of polymorphonuclear leukocytes. Haserick[8] feels that the clumping of the polymorphonuclear leukocytes in a rosette pattern probably precedes the development of the "L.E." cell. It is his belief that this clumping should be considered as diagnostic as is the final phase (the "L.E." cell) of this phenomenon. Plasma from patients with chronic discoid lupus erythematosus and other diseases failed to induce this phenomenon.

It, therefore, becomes increasingly apparent that the phenomenon of the "L.E." cell originally described by Hargraves is specific for acute disseminated lupus erythematosus. Its value as a diagnostic aid has been well substantiated. Its value in opening new fields of investigation for the etiology and therapy of this disease is self-evident.

REPORT OF CASES

Case 1. History. Mrs. M. E. J., 38 year old white housewife, was admitted to the Sheltering Arms Hospital on July 11, 1949. She complained of a skin rash on the face and other exposed areas of the body. Fever and fatigue had been present for about two months, and migratory arthritis had been noted for nine months preceding this admission.

Because of this episode and similar ones that had occurred regularly every spring and summer for the past thirteen years she had seen many physicians and was diagnosed as having pellagra. In spite of specific vitamin therapy the condition was not cured, but invariably would "clear up" in the fall only to return the following spring. An allergy of the skin to sunlight was also considered to be a factor. The past history, family history and systemic review were essentially non-contributory.

Physical Examination: Positive findings on admission were: Temperature 100°F., the pulse rate 72 per minute, and the respirations 20 per minute. The blood pressure measured 120 mm. Hg. systolic, and 80 diastolic. The skin showed a diffuse, red maculopapular eruption with areas of dry, scaly exfoliation. There was no exudate. The mouth showed gingivitis and marked dental caries. The heart and lungs were grossly negative, as was the abdomen.

Laboratory Findings: Repeated urinalyses showed albuminuria, pyuria, and on rare occasions microscopic hematuria. The hemoglobin varied from 86 per cent on admission to 56 per cent on October 19, 1949, while the erythrocytes on these occasions were 4.5 and 2.9 million per cu. mm., respectively. The total leukocyte count on admission was 11,750 per cu. mm. with a differential count of 62 per cent neutrophils; 12 per cent eosinophiles, 21 per cent lymphocytes and 5 per cent monocytes. Subsequent counts were within normal limits or were of definitely leukopenic values. The sedimentation rate on two occasions was 30 and 32 millimeters in one hour. Blood non-protein-nitrogen measured 27 mgm. per cent, while the total proteins were 7.0 mgm., per cent, albumin 3.5 grams per cent and globulin 3.5 grams per cent. A skin biopsy was made soon after admission, but the findings were not thought to be diagnostic of any specific disease. An electrocardiogram was within normal limits on admission. A roentgenogram of the chest showed "prominent hilar shadows and increased bronchial markings to the right base."

Course in the Hospital: This patient remained ill with an erratic clinical course. She was in extremis several times, only to improve to the point that a remission seemed likely. In addition to migratory arthritis, there developed pericardial and pleural effusion, cardiac dilatation and marked alopecia. At no time have her dermatologic lesions cleared. Because of anemia and leukopenia, many transfusions of whole blood were given. At the present writing she is home, doing her housework. The hair is returning and her symptoms have practically abated.

Hematologic Studies: July 20, 1949: Sternal marrow aspiration:

Concentrated marrow: The marrow is hyperplastic. There is some left shift in the myeloid cells and toxic granulations are seen. Erythropoiesis is normoblastic and normal. Platelets seem adequate.

There are many mature polymorphonuclear leukocytes which have engulfed a purplish homogenous mass. These have the same characteristics of the "L.E." cell described by Hargraves, who personally confirmed these findings.[4]

August 6, 1949: Concentrated marrow: This marrow is slightly hyperplastic. The granulocytes show marked toxic changes. There is vacuolization of the cytoplasm of many granulocytes. Also, there is found a *rare* polymorphonuclear cell that has a homogenous, purple inclusion body that has pushed the nuclear lobes to the periphery. This is probably an "L.E." cell and the sparcity of them probably indicates a remission.

September 1, 1949: Concentrated, heparinized venous blood preparation. This slide shows numerous stages of nucleolysis in the toxic polymorphonuclear cells. In addition, there are many cells with the typical characteristics of "L.E." cells.

June 10, 1950: L.E. cells found on admixture of patient's plasma with "buffy coat" of heparinized venous blood of normal individual, according to technique of Moffatt and co-workers[9].

Conclusions: Acute disseminated lupus erythematosus was established as the diagnosis on the basis of these findings in the blood and marrow. Hargraves[4] supported this diagnosis as did Haserick[8], who, independently caused the "L.E." phenomenon to appear in normal marrow with plasma from this patient.

The patient has run a typical course on this admission with the cardinal points of the disease being illustrated. She, at present writing, appears to have gone into a clinical remission, despite the fact that there is hematologic evidence of activity still present.

Case 2. History. E.W.P., a sixty year old white male, was admitted to St. Elizabeth's Hospital February 14, 1950, complaining of chest pain, chills, and fever. More than ten years previously, following the use of sulfanilamide for rheumatism, he had become "deathly sick" and he had had a fever for about one year. Since then he occasionally has had mild transient arthralgia. Ten years ago he received x-ray therapy for a rash on his face. He developed pneumonia three years ago, and had an uneventful recovery.

He remained well until about December 1, 1949, when he developed an upper respiratory infection, anorexia, weight loss, malaise, post-prandial epigastric distress and flatulence. His symptoms continued and on January 12, 1950, he was admitted to a hospital in another city where he was found to have fever, elevated blood pressure, and pyuria. The pyuria cleared and he returned home in ten days. His condition gradually declined, and after two weeks, he developed intermittent pain and swelling of the hands, feet and shoulders, lasting two to three days each time. Three days prior to admission to the hospital he developed sharp left shoulder pain, radiating down the anterior left chest to the left costal border. The pain was more severe on deep inspiration. He had no cough or hemoptysis. He then began having chills and fever.

Physical Examination: Positive findings on admission were: Temperature 103.8°F., pulse rate 110 per minute, and respiration 26 per minute. The blood pressure measured 160 mm. Hg. systolic and 90 diastolic. The patient was restless and appeared to be acutely ill. The lungs were clear to percussion and auscultation, and the remainder of the physical examination was essentially negative.

Laboratory Findings: The Mazzini test was negative. The hemoglobin was 10 grams per cent and the erythrocyte count was 3,300,000 per cu. mm. The leukocyte count was 5,850 per cu. mm. with 72% neutrophils, 27% lymphocytes and 1% monocytes. On one occasion the leukocyte count rose to 12,200 per cu. mm. However, all other blood counts showed a persistent leukopenia and anemia. The platelet count was 119,000 per cu. mm. Urinalysis revealed a 3 plus albumin, 4-8 WBC and many RBC per high power field. Transient hematuria was seen on repeated urinalyses. The NPN was 30 mg. per cent. There was 40% retention of PSP in two hours. Blood cultures were negative. All agglutinations, including the Rickettsial group, were negative. Pseudomonas and E. Coli were cultured from the urine. Feces cultures were negative. Roentgenogram of the chest on admission showed increased broncho-vascular markings, elevation of the left diaphragm and an area of infiltration at the left base. This gradually cleared on subsequent films. Intravenous pyelograms were negative. The electrocardiogram was normal.

Course in Hospital. On cystoscopic examination the bladder mucosa was hyperemic. RBC were present in the urine from both kidneys. On the seventh hospital day the patient developed a hemorrhagic "butterfly" rash on the cheeks and across the bridge

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of the nose. He complained of generalized aching and appeared to be acutely ill. By the tenth day the rash had spread to the exposed upper portion of the chest. Two days later the rash had become generalized and was hemorrhagic and confluent, and faded on pressure. At this time the facial rash began to fade and become crusted. On the fifteenth hospital day, the rash and systemic symptoms began to subside and he gradually and steadily improved.

Hematologic Studies: Unconcentrated marrow: Hypoplastic marrow with normoblastic erythropoiesis somewhat depressed. The granulocytes are left shifted but not abnormally. There is an increase in platelets and the number of reticuloendothelial and plasma cells.

Concentrated marrow (heparinized): This preparation is literally "loaded" with cells that have all of the characteristics of the "L.E." cell described by Hargraves. From a hematologic standpoint, the diagnosis of disseminated lupus erythematosus could be made from this study.

Impression: Marrow findings consistent with clinical diagnosis of acute disseminated lupus erythematosus.

Case 3. History.—N. W., a twenty-two year old Negro male, was admitted to the Veterans Administration Hospital* for the first time in March, 1947. Except for three attacks of gonorrhea, he had always been in good health until March, 1946, when he began having a transient feeling of fullness in the right anterior side of the chest. In December, 1946, he began having occasional sharp pains in the right chest, mild exertional dyspnea, and a questionable increase in cough. He had no known fever or other symptoms, but the chest pain and dyspnea persisted and he came to the hospital. He was found to have a right middle lobe pneumonitis, which cleared to x-ray and physical examination without any specific therapy. The leukocyte count was 4,800 per cu. mm. with a normal differential leukocyte count. The hemoglobin was 11.3 grams per cent and the RBC 4,060,000 per cu. mm. The Kahn test was negative. There was a trace of albumin in the urine on two occasions. Following discharge he felt well until March, 1947, when he developed arthralgia of the shoulders, elbows, knees and ankles a few days following a mild upper respiratory infection. He soon developed slight swelling of the lower left leg and returned to the hospital. There were a few pigmented scarred areas on both cheeks. There were small, firm, non-tender, cervical, axillary, epitrochlear, and inguinal lymph nodes. The lower left leg had slight pitting edema and there was pain on motion of shoulders, elbows and knees. Otherwise, physical examination was not remarkable. He again had anemia, leukopenia and slight albuminuria. Total proteins were 8.8 grams per cent, with albumin 3.6 and globulin 5.2 grams per cent. There was no sickling of the erythrocytes. Agglutination studies, including test for Brucellosis, were all negative on two occasions. Sternal marrow examination and roentgenogram of the chest were reported as being normal. A biopsy of an epitrochlear node was reported as reactive lymph node. The patient ran a daily low-grade fever during the three months of hospitalization. He gradually improved symptomatically and was discharged. Since that time he has been seen frequently and has been studied exhaustively on several occasions. He has continued to have essentially the same complaints and physical and laboratory findings. He twice developed a severe anemia, requiring several transfusions of whole blood each time. Lymph node biopsy again was reported as "reactive lymph nodes." Muscle biopsy showed many of the blood vessels to be surrounded by dense foci of lymphocytic infiltration. Blood cultures were all negative. Many electrocardiograms were made, and all were considered to be normal. He was given para-amino-benzoic acid, 1.2 grams daily, for several months without any definite improvement. In November, 1948, the patient had a generalized convulsion, lasting five minutes, at which time he bit his tongue. Two electroencephalograms were normal. Neurologic examination was negative and examination of the spinal fluid was normal. The patient continued to have infrequent grand mal seizures. At times he appeared very lethargic. The electroencephalogram in June, 1949, was "grossly abnormal, showing a S-2 record and spike activity in all leads, indicating epilepsy and allied disorders." He was started on dilantin, 0.1 grams three times daily, which he has continued to take, with a decrease in the number of seizures. In December, 1949, he again began having increased pain in his chest and peripheral joints and swelling of the ankles and feet.

*Reported with the permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

Six days later he had two grand mal seizures one hour apart and was brought to the hospital.

Physical Examination: Positive findings were: Temperature 102° F., pulse rate 110 per minute and respirations 22 per minute. The blood pressure measured 125 mm. of Hg. systolic and 80 mm. diastolic. There were small pigmented areas over the malar eminences and on the forehead. The apical impulse was 3 cm. outside the mid-clavicular line in the fifth left intercostal space. The heart and lungs were otherwise normal to percussion and auscultation. The liver edge was smooth and palpable 2 cm. below the right costal margin. Moderate pitting edema of both feet was present. The left elbow was slightly swollen. There was moderate generalized lymphadenopathy. Neurological examination was negative.

Laboratory Studies: The cardiolipin test for syphilis was negative. The hemoglobin was 12.2 grams per cent and the leukocyte count was 6,000 per cu. mm. with 54% neutrophils and 42% lymphocytes, 2% monocytes and 2% eosinophils. The leukocyte count ranged between 3,500 and 8,000 per cu. mm., usually being less than 5,000. Many urinalyses were negative except for a trace of albumin on three occasions and 1-2 RBC per high power field on one occasion. The total proteins were 8.9 grams per cent with 3.4 grams albumin and 5.5 grams globulin. With the onset of jaundice, the serum bilirubin rose to 6.4 mg. per cent, 3.4 mg. direct and 3.0 mg. indirect. The thymol turbidity was 26 and the cephalin flocculation was 4 plus in 24 hours and in 48 hours. The urine urobilinogen was 1.3 mg. (two hours specimen). An electroencephalogram was very abnormal, indicating focal abnormality in the frontals and generalized disturbance of cerebral physiology. Roentgenogram of the chest showed an increase in the transverse diameter of the heart in comparison with previous films, but was otherwise normal. Barium meal examination was normal.

Sternal Marrow Examination: Examination of several concentrated heparinized bone marrow smears showed a moderate number of polymorphonuclear leukocytes which had engulfed homogeneous blue to purplish material. These had the characteristics of the "L.E." cell described by Hargraves. The marrow showed myeloid hyperplasia and normoblastic erythropoiesis.

The impression was that this concentrated specimen substantiated the clinical diagnosis of disseminated lupus erythematosus.

Course in the Hospital: About one week after admission the patient developed malaise, anorexia, right upper abdominal discomfort, hepatomegaly, icteric sclerae, dark urine, and abnormal liver function tests and it was thought that he had homologous serum jaundice. He had an uneventful recovery in one month. On February 6, 1950, examination of concentrated smears of heparinized bone marrow revealed typical "L.E." cells. Since then the patient has gradually improved and is relatively asymptomatic. However, he still has his original complaints, i.e., a feeling of fullness in the right chest, exertional dyspnea, and intermittent moderate pain, stiffness and swelling of various peripheral joints.

Case 4. History.—M.E.C., 18 year old white female, noted eruption involving face September, 1948. Shortly thereafter there was anorexia, nausea, vomiting and fever. The patient was hospitalized for one week in November, 1948. During this period a specimen of skin was removed for biopsy which on examination was suggestive of acute disseminated lupus erythematosus. Within a short time after discharge from the hospital the patient lost most of her hair.

In March, 1949, the patient was given sodium para-amino-benzoic acid. This was followed by a generalized maculo-papular eruption. This rash cleared promptly after the withdrawal of this drug.

The patient was able to continue with her school work until March, 1950. Anorexia, fever, pain in the joints and general weakness were the prominent complaints. The patient was admitted to the hospital. The physical findings at that time were a generalized maculo-papular eruption most prominent over the malar eminences. There were no other findings of note.

Laboratory examination included numerous normal urinalyses. The total white count varied between 2650 and 4000 per cu. mm. Because of severe anemia a number of transfusions were given. The "L.E." cell was demonstrated in a concentrated sternal marrow aspiration in April, 1950.

Case 5. History.—Mrs. H.N.T., a 39 year old white female, has had intermittent pain and swelling of various joints since 1946. It was thought that she had either rheumatic fever or rheumatoid arthritis, and she was given two courses of chrysotherapy

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without improvement. The patient had a recurrence in March, 1950, and was found to have a soft systolic apical murmur in addition to the joint manifestations.

The erythrocyte count was 4,200,000 per cu. mm., the hemoglobin 85%, and the leukocyte count 4,800 per cu. mm. Examination of the urine revealed a trace of albumin and microscopic hematuria. She began running an intermittent fever, the temperature reaching 104°, the murmur became more intense, and she was thought to have subacute bacterial endocarditis. All blood cultures were negative. Electrocardiograms were negative. She received large doses of penicillin without improvement. On May 24, 1950, "L.E." cells were found in the myeloid-erythroid layer of concentrated heparinized sternal marrow. At no time has this patient had any cutaneous lesions, and she may be considered as a case of lupus sine lupus.

Case 6. History.—Mrs. S.H., aged 52, had been treated for hypertension for a number of years and had no other complaints until March, 1950, when she developed a skin eruption on her face. This was described as a well defined, erythematous, annular rash, believed to be most suggestive of a drug eruption.

She was hospitalized with cardiac dilatation and a blood pressure of 190 mm. Hg. systolic, and 90 diastolic. There was low grade fever up to 100.6° F. Because of the question of disseminated lupus erythematosus, aspiration of the sternal marrow was performed on March 27 and the heparinized concentrated preparation was reported as: "only a rare cell characteristic of the L.E. phenomenon could be found. There were many neutrophils, however, where large vacuoles with nuclei pushed to the periphery around them—suggesting that the homogenous inclusion body had been lost. Finding only a rare characteristic L.E. cell suggests that this is acute disseminated lupus erythematosus in remission."

At present, her lesions have practically cleared in a three months' period and there is no physical discomfort.

Case 7. History—Mrs. K.E.McE., a 38 year old housewife, became sunburned while working in her garden in June, 1949. The erythema did not clear on her face, but instead spread from the bridge of the nose to the forehead and malar prominences accompanied by moderate edema. There was no particular sensation to this rash, but it progressed to involve the arms and legs. Fatigue and shortness of breath were the only other complaints.

Two months after the onset, she was hospitalized. A moderate hypochromic anemia and leukopenia were noted. The electrocardiogram showed minor T wave changes. Her temperature ranged from 100° to as high as 105° F. In August, 1950, concentrated heparinized marrow obtained from the sternum on two occasions showed only a rare neutrophil with homogeneous inclusion bodies and nuclear lobes pushed to the periphery. The diagnosis of disseminated lupus erythematosus was suggested.

In eight months her lesions cleared markedly, but of interest is that a repeat marrow aspiration in April, 1950, showed more than twelve typical neutrophils with the "L.E. phenomena" on the concentrated specimen. Easy fatigability and dyspnea on exertion were her chief complaints. It was felt that this patient definitely has disseminated lupus erythematosus, but it has been quiescent in recent months.

SUMMARY

The clinical and laboratory manifestations of acute disseminated lupus erythematosus have been briefly reviewed.

The "L.E." cell found in preparations of the bone marrow and peripheral blood in patients with acute disseminated lupus erythematosus has been reviewed and its significance discussed. That it may be related to an immunologic response by the body is supported by the finding that plasma of these patients incubated with normal bone marrow induces the formation of these cells.

Seven cases are cited in which the diagnosis of acute disseminated lupus erythematosus was made by demonstrating the "L.E." cell in bone marrow or peripheral blood. Three cases are presented in detail.

CONCLUSIONS

1. The "L.E." cell is a diagnostic aid in acute disseminated lupus erythematosus.

2. The "L.E." cell may usually be found in bone marrow or peripheral blood preparations if proper care is taken in obtaining such material.

3. The "L.E." cell may be induced by incubating normal bone marrow or venous blood with plasma from patients with acute disseminated lupus erythematosus.

Acknowledgement.—We wish to thank the following physicians for permitting us to report five of the seven cases presented: Douglas G. Chapman, T. Dewey Davis and Thomas W. Murrell, Jr.

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Cervix Examinations Urged for Early Cancer Detection.

A woman who has an ideal cancer detection examination of the cervix with no abnormal findings may be reasonably sure that no clinically important cervical cancer will develop within six months, and perhaps longer. This opinion was expressed by Dr. Howard W. Jones, Jr., Baltimore gynecologist, writing in the July 28 *J.A.M.A.*

Dr. Jones said an "ideal" examination should consist of a smear study, microscopic examination of tissue removed from an abnormal tissue, feeling and a history-taking.

He pointed out that cancer of the cervix is responsible for about one out of every seven female cancer deaths. Cervix cancer, he said, in many instances has an insidious development and when symptoms appear the disease is far advanced.

Consequently, he stressed the importance of smear and biopsy studies in cases of abnormal cervices where no symptoms have developed. Inspection alone is not a reliable diagnostic method for small cancers, he said.

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AN EVALUATION OF THE EPILEPSY PROGRAM IN VIRGINIA*

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There are approximately five hundred thousand epileptics in the United States. This figure is of course only an estimate. So long as epilepsy remains difficult to define exact census figures will not be possible. Authoritative reviews of the problem, however, reveal that the statistical incidence of epilepsy is somewhere between four and six per thousand population. It is interesting to note that agricultural areas have higher epileptic "rates" and also regions with a high percentage Negro population. Negroes are generally found to be twice as susceptible to epilepsy as other races.

On the basis of the above considerations (and Virginia's population of over three million—22% Negro) it would not be unreasonable to estimate that our Commonwealth has 15,000 epileptics.

Since epilepsy is statistically as common as tuberculosis or diabetes, the general practitioner should be at least as familiar with the diagnosis and treatment of epilepsy as he is with these other "more popular" diseases, and as informed as to what is being done and can be done for them in Virginia.

It is obvious from these large figures that only a very tiny percentage of the epileptics in Virginia are being cared for by public health agencies. The remainder of the epileptics (who are getting any medical care at all) are certainly being treated by the family doctor or private specialists.

First, we will review the state institutional facilities for those severe epileptics whose sickness is such that they require intramural care. The Lynchburg Colony for epileptics and feeble-minded patients was opened in 1911 and now has an average epileptic population of around 500. It is the most important epileptic center in the State. Conditions in this hospital are hopelessly overcrowded. Unless an epileptic is of school age (and therefore considered eligible for training) or unless he has become a menace to public health, it is likely that he will be placed on a waiting list and will not be able to obtain hospitalization in Lynchburg. Of course, epileptics who have a psychosis in addition to their epilepsy, may be hospitalized as "insane" in the regional hospitals at Marion, Staunton, Williamsburg and Petersburg, the latter hospital caring for the Negro epileptics (in a department separate from the psychotic epileptics).

Although nearly twice as many Negroes as white subjects suffer from epilepsy, there is an outstanding discrepancy in the figures for Lynchburg and Petersburg. Approximately 500 whites are cared for in the Colony at Lynchburg but the average non-psychotic epileptic population in Petersburg is around 60. It is presumed that if there were proper facilities that a much higher number of Negro epileptics could and would be hospitalized. Actually, there are not sufficient facilities in either of these two designated hospitals for Virginia's epileptics in need of intra-mural care.

Although there is no exact knowledge available, it is not likely that any sizable number of epileptics are maintained in private hospitals in Virginia.

Before leaving the subject of the intra-mural care in Virginia it must be noted that the Commonwealth has had, for approximately 25 years, a law for the eugenic sterilization of epileptics and feeble-minded individuals and this law has been applied cautiously in some cases where there was a strong familial tendency toward reproduction of epileptics and mental defectives. Indeed it was the decision of Supreme Court Justice Oliver Wendell Holmes in the case of Buck vs. Commonwealth of Virginia that led to the first practical eugenic sterilization procedure in the United States.

Extra-mural care for Virginia epileptics is a somewhat more hopeful matter to report. Both Federal and State government has provided facilities for such care, but the largest single effort in Virginia is provided by the "Virginia Society for Crippled Children and Adults" at the University of Virginia

*Presented at meeting of the American Branch of International League Against Epilepsy in Cincinnati, Ohio, April 15, 1950.

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Hospital in Charlottesville. This anti-convulsive clinic was utilized by two thousand three hundred and thirty-eight persons from January, 1945 (the date at which the clinic was set up), until April 1, 1950. It is sponsored by the University of Virginia Hospital and has field clinics in various parts of the State where their services are most needed. It gives an opportunity for complete medical, neurological and psychiatric examinations, psychological testing when indicated, electroencephalogram when necessary and arranges for hospitalization or special tests when an indication for them is found. The field clinic operates by arrangements made in advance. This division is headed by Drs. Leigh and Klingman of the University of Virginia Hospital. Clinics are held either in areas where the most cases are found, or where there is the greatest need.

For example, the Richmond area is not included because of the availability in the Richmond area of the Medical College of Virginia clinic. At the present time eight clinics are held in the following order and areas: March—Norfolk and Newport News; April—Norton; May—Bristol and Wytheville; September—Winchester and Arlington; November—Danville. These clinics are scheduled in advance with the permission of the local health officer. Arrangements are made as to the place to hold the clinic, usually through the local Health Department, or if held in a hospital, through the superintendents of the local hospital. The secretary of the Anti-convulsive Clinic visits the area in question a week prior to the date set and contacts local organizations and physicians who might care to refer patients. Organizations particularly interested in referring patients have been the Department of Public Welfare, Child Welfare Agencies, Health Departments and Social Service Agencies. There is no charge made to the patient or the referring agency for the visit. Results have been reported as very gratifying. Local medical societies and physicians have been cooperative and willing to accept help in that field of work which they feel they do not understand too well. Results from therapy have also been good. It is estimated that 25% of the patients have obtained good control of their seizures, either with complete cessation or marked reduction in the number of severity. This success would suggest the possibility that if institutionalized cases had the advantage of intensive care as provided by the Society for Crippled Children that quite a few might not need to remain always in the hospital. Fifty per cent of the outpatients in this clinic have obtained "fair" results, although still having a sufficient number of seizures to present a problem. The remaining 25% have obtained little or no relief and in these cases there is usually severe organic damage or deterioration, cases which are usually classified as "hopeless." For the first two groups a contact has been established with the State Rehabilitation Service, an agency most helpful in attempting rehabilitation of such patients.

The outstanding difficulties that this clinic has encountered have been first an indifference on the part of the patients to report their progress, and secondly, the lack of sufficient personnel, such as a Social Service worker who could look into the homes of these patients and maintain close contact with those patients who need to be urged to regularly continue their medication. Efforts need to be made to have some qualified person to visit the school, talk to the superintendent so that the epileptic children who are frequently barred, would be allowed to get a normal education and be treated as normal children insofar as possible.

One of the values of the epilepsy program in this clinic has been the opportunity to evaluate new medications prior to their general release. Thiantoin of Eli Lilly Company was on trial for a period of about two years in this clinic along with other clinics throughout the country before it was put on the market. Phenurone is now under investigation.

Less extensive extra-mural or clinic treatment care is furnished by the Medical College of Virginia Clinic, headed by Dr. P. H. Drewry and under the direction of the Department of Neuropsychiatry at the Medical College of Virginia. Dr. Drewry is assisted by a large staff. Here, twice weekly, patients are examined and furnished with drugs for the control of their epilepsy. When indicated electroencephalography is available and, of course, all the diagnostic facilities of the Medical College of Virginia are available to the patients in this clinic. In addition, there are available consultations with other specialists in neurology, neurosurgery, and internal medicine. This clinic treats over a hundred new epileptics during each fiscal year. The total annual number of epileptic *visits* to this clinic has not yet been estimated.

The Federal agencies located in Virginia (par-

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ticularly the Veterans Administration) have a sizable contribution in the total picture. A small out-patient department is operated in the Richmond area by Dr. Wilfong and Dr. Kanich. However, under present laws veterans whose epilepsy is not service connected are not eligible for out patient treatment for this condition from the Veterans Administration. McGuire Veterans Administration Hospital furnishes diagnostic and consultation service for veterans who develop epilepsy and short term hospitalization can usually be provided for a veteran whose epilepsy is new, or requires hospital study (when beds are available). The hospital in Roanoke, Virginia, of course, cares for service connected epileptics who have psychoses.

The general hospital in Richmond (McGuire) has been designated as one of the three national epileptic "centers" and, accordingly, a large number of epileptic patients are treated and studied here. The department which handles the problem is under the direction of Dr. Benedict Nagler, assisted by Dr. James B. Funkhouser. In addition to the study of epileptic veterans, a training program for technicians and physicians in electroencephalography is given at McGuire Veterans Administration Hospital.

An insignificant number of epileptics, of course, is provided domiciliary care in the soldier's home at Kecoughtan, Virginia.

It can be readily seen that by far the largest number of epileptics in Virginia are treated by the general practitioner and by certain specialists as private cases. It is not the purpose of this paper to discuss these cases. There is no doubt that patients who can afford private medical care will get much better attention from private doctors because the public facilities, both Federal and State, are extremely meagre. It is not the intent of any of the above mentioned State or Federal agencies to compete with the excellent private management of epileptic patients whenever private care is financially possible for the individual and his family.

An analysis of the problems encountered in the care of epileptics has been made by the authors and will be briefly discussed here. First, there is considerable difficulty encountered in integrating the above agencies because of duplication of effort, lack of complete statistical reporting, differences in terminology. For example: Some clinics and institutions used the word "epilepsy" and others "convulsive disorder." (For the same clinic—the Medical College of Virginia—the State Board of Mental Hygiene and Hospitals listed only 72 new cases during the same fiscal year that the Medical College of Virginia reported 106 epileptics.)

The name "epilepsy" is unfortunately associated with stigma. Well meaning people have attempted to make popular the name "convulsive disorder" in order to "de-stigmatize" the epileptic problem. However, petit mal, psychomotor and akinetic attacks, are epileptic in character, though not convulsive. There are no clinically observable convulsions incident to these latter diseases. Therefore, many of these other epilepsies are not reported as convulsive disorders.

Another difficulty frequently encountered is in obtaining liaison with the practicing physician. As reported, the anti-convulsive clinic at the University of Virginia has made the best effort in this direction. The Veterans Administration Hospital in Richmond is able to give complete summaries in each case if the epileptic is willing to sign consent for the release of information to the family physician and summaries are forwarded to the family physician when the patient is willing for this information to be released. The Medical College of Virginia, of course, makes reports to the referring physicians (with the patient's consent).

However, it is our experience that most physicians are not aware of the importance and magnitude of the problem and are indifferent to the problem of the epileptic. The social agencies in many instances show a lack of interest and an attitude of hopelessness toward the problem, possibly because of their continued failure in their efforts to convince employers that epileptics can be rehabilitated and are industrially useful.

High on the list of difficulties encountered in the treatment of epileptics by the above described facilities is the lack of funds for the provision of medication to "out-patients". The State institutions, of course, are seriously hampered and crippled by the insufficient amount of money provided for their intra-mural epileptics and there is no way for the epileptic to have medication after he leaves the hospital. The same difficulty in obtaining extra-mural treatment pertains in the Veterans Administration to non-service connected veteran epileptics.

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Probably the greatest difficulty in the care of epileptics (not in a hospital) is the matter of cooperation with employers. Employers are very loath to hire epileptics and our most frequently heard plea from our patients is "do not tell my boss I have epilepsy or I will lose my job." This is not always the fault of the employer. It may be the co-workers who object to working along side an epileptic who may have seizures. Certain organizations, such as the "Good Will Industries" have tried to prove their practical employability by direct facts. However, even in a recently published book of Ruskin Taylor's "New Hope for the Handicapped", the social problem of the epileptic's vocational readjustment is discussed only in a few words. Organizations such as the International League against Epilepsy, American Epilepsy League and several good lay organizations are working on this tremendous problem with some recent success.

Plans have been made in Virginia which may help solve a great many of these problems detailed above. An increase in hospital facilities for Negro patients is already approved in a development at Elko, Virginia.

In a recent report to the Governor by the Duke Commission it was recommended that a centrally located "Institute for Epilepsy" (with departments for white and Negro patients) be set up in cooperation with the Department of Neurology and Psychiatry of the University of Virginia. This would be a large step in the direction of integrating the state's scattered efforts in behalf of the epileptics. It might be possible that this institute could take the initiative in establishing a closer cooperation between clinics, hospitals and institutions, both Federal and State and private, thus avoiding duplication of effort. Such an Institute could set up an educational program to arouse and maintain interest in the social problems of the epileptics. It is certain that a centralized reporting of epilepsy would make possible a better statistical survey and promote the exchange of material, which would be of mutual advantage to all interested agencies. Although some progress must wait until the various theories and "schools" of epilepsy have been solidified as to terminology and therapy, a central institute could certainly be instrumental in helping to establish a definite classification and treatment routine.

Progress has been made in the integration of the efforts of the State Board of Mental Hygiene and Hospitals, the medical colleges and the Veterans Administration hospitals, as evidenced by this paper. However, much more can be accomplished in this field.

In conclusion, the outlook for the epileptic in Virginia is, despite all the disadvantages cited, very good. New advances in diagnosis, better drugs, and a wider knowledge on the part of the doctors themselves in the treatment of this condition in the past decade, have caused the epileptic to suffer fewer and less severe convulsions. It is not too optimistic to hope that the sustained effort of all who are working in this field, both layman and doctor, may eventually cause the general public to lose its superstitious fear of "the sacred disease". When this is accomplished a magnificent goal will be won.

A.M.A. Supports Bill Banning Sale of Fireworks.

The American Medical Association has filed a statement with the House Judiciary Committee supporting its bills restricting the sale of fireworks.

The statement said that the medical profession for many years "has been concerned with the serious menace to life and health resulting from the use of fireworks." It added that the Association is in favor of proposals such as the bills under consideration, "which are designed to alleviate health hazards of this type."

The bills would prohibit the shipment of fireworks into any state or political subdivision in which their sale is prohibited, unless intended for public displays or other purposes specifically authorized by law.

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ENDOMETRIAL HYPERPLASIA IN ELDERLY WOMEN WITH HEPATIC CIRRHOSIS*

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It is well known that in women during the reproductive period one of the functions of the liver is to destroy or excrete estrogens, and that this function may fail in chronic liver disease, resulting in various manifestations of hyperestrinism.

Among our routine autopsies, however, I recently saw two cases of chronic liver disease associated with endometrial hyperplasia in elderly women of 69 and 71 years of age. This prompted me to investigate other cases of chronic liver disease in elderly women.

The enquiry was limited to women over 60 years of age. Unfortunately in many of the autopsies performed previous to 1940 routine sections of endometrium were not made. We found however 16 cases suitable for examination (Group B). For comparison with these, 16 control cases were also examined (Group A) consisting of women of similar ages but without known liver disease.

TABLE I

SHOWING THE ASSOCIATION OF ENDOMETRIAL HYPERPLASIA WITH CHRONIC LIVER DISEASE IN ELDERLY WOMEN

	Group A (Controls)	*Group B* (Hepatic cirrhosis)
1. Numbers in age groups:		
60-64	2	2
65-69	9	9
70-74	5	5
Total	16	16
2. Types of liver cirrhosis	None	Portal 6
		Cardiac 5
		Biliary 5
3. Condition of Endometrium		
Normal (senile atrophy)	7	1
Hyperplasia +	6	3
Hyperplasia ++	3	5
Hyperplasia +++	0	7

The results are set out in Table I. The cirrhoses found were of the portal, cardiac and biliary types. For purposes of comparison the endometria were divided into four groups, i.e., normal (senile atrophy at the ages of these patients) and hyperplasia of three degrees, indicated by the number of plus signs. It will be seen that whereas in the control group there are seven normal endometria, in the cirrhosis group there is only one. The cirrhosis group, however, contains seven high grade hyperplasic endometria, but the control group none.

Clinically, neither bleeding nor other gynecological manifestation was noted in any of the women with cirrhosis and endometrial hyperplasia.

In the light of present knowledge these facts would seem to indicate a relative hyperestrinism in the cirrhosis group.

Since estrogens are secreted by both the ovary and the adrenal cortex, and the former would appear to be excluded in women over 60 years of age, it seems that the adrenal cortex continues to secrete estrogens in women even into old age, but that the liver normally keeps these hormones at levels too low to produce known physiological effects. In certain forms of liver disease, however, this regulatory action is lost and some degree of endometrial hyperplasia occurs, but without clinical manifestations.

*From the Department of Pathology, Medical College of Virginia, Richmond.

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Medical Society of Virginia Cancer Committee

Chairman, GEORGE COOPER, JR., M. D.

University, Virginia

Reprints of this and preceding Bulletins may be obtained from this office

November, 1951

Cancer of the Lung

Primary pulmonary malignancy is on the increase. Since chronic irritation is a known factor in carcinogenesis, probable explanations are that a constantly higher percentage of our population lives in the smoky, dusty air of cities and that the use of cigarettes is increasing. In Virginia in 1930, 42 deaths from cancer of the lung were reported, in 1950, 103. Most of the victims are men over 50, but women and the younger age groups are not immune. Similar increases, too great to be explained as due to more accurate diagnosis, are reported throughout the country.

Until 1933, primary pulmonary carcinoma was a disease which physicians had no means of curing. In that year, Graham and Rienhoff individually performed successful total pneumonectomies for carcinoma. During the intervening years, hundreds of people, most of them victims of cancer of the lung, have survived the operation. Improvements in technique, in combatting shock, and developments in anaesthesia and chemotherapy, have reduced the mortality rate from around thirty per cent to the neighborhood of twelve per cent. Old age is not a contraindication to the procedure which has been successfully carried out on patients in the late sixties. The compensatory power of the remaining lung has been found to be so great that loss of a lung has proven a minor physical handicap, disturbing to professional athletes only. Nor is the operation usually deforming, the thoracic cage remaining intact because, in the absence of complications, thoracoplasty is unnecessary.

As to the value of the operation in pulmonary carcinoma, the end results of a method of treating cancer cannot be accurately stated on the basis of only eighteen years experience, particularly since few of the total pneumonectomies were done in the first part of the eighteen year period. However, the following statements cannot be disputed:

1. A number of people are living and apparently free of disease five years after total pneumonectomy for primary cancer of the lung, and a few are living and well ten years or more after the operation. **The possibility of cure of a hitherto incurable disease has been established.**

2. These tumors are uniformly radioresistant. **Pulmonary resection, preferably total pneumonectomy, including removal of the bronchial and tracheal nodes on the affected side, offers the only possibility of cure.**

3. If a cure is to be attempted, **the patient must come to surgery before the cancer has involved the pleura and before it has metastasized beyond the bronchial and tracheal nodes on the affected side.**

4. The operative mortality rate compares favorably with that of most major surgical procedures, and is steadily decreasing.

In view of these facts, it is of vital importance that laymen be instructed in the early symptoms of primary pulmonary malignancy so that they will seek medical advice early; and it is of even greater importance that physicians refer suspicious cases to a competent thoracic surgeon while the disease is still in an operable stage.

Unfortunately, cancer of the lung produces no characteristic clinical picture. The most frequent cause of delay, and almost invariably a fatal delay, in diagnosis of cancer of the lung is the making of a clinical diagnosis, without careful investigation, of tuberculosis, lung abscess, unresolved pneumonia, bronchiectasis, or heart disease.

Early Symptoms:

Cough — The majority of adults suffer to some degree from a chronic cough. When the cough deviates from normal for the individual, immediate investigation is indicated. In seventy to seventy-five per cent of pulmonary cancer, unusual cough is an early symptom.

Hemoptysis — In about sixty to sixty-five per cent of cases, hemoptysis, varying from streaking to copious expectoration, is an early symptom. Occasionally tuberculosis and cancer occur in the same lung. Even in the presence of a few tubercle bacilli in the sputum, in the absence of conclusive x-ray evidence of tuberculosis or bronchiectasis, hemoptysis should be considered as due to an intrabronchial growth, and this probability immediately investigated.

Pain — Occurring in about fifty per cent as an early symptom, pain from cancer of the lung is a constant, deep, dull ache, usually at

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the level of the tumor. It is presumed to be due to pressure by the tumor on the bronchial wall. It is not a pleuritic pain, which is associated with the respiratory cycle, and of course, pain due to invasion of the chest wall and brachial plexus is a late manifestation.

Loss of Weight — In about forty per cent of cases, there is an early, striking loss of weight, more than can be explained by cough and hemoptysis, and the worry incident thereto. In these cases, following pneumonectomy, though only a relatively small growth is present, there is a prompt, striking gain in weight.

Hyperpnea — About twenty-five per cent of patients experience a sudden paroxysmal desire to breathe deeply, almost an air hunger. It lasts from a few seconds to a few hours. A possible explanation is that a bronchus partially occluded by a tumor is suddenly further occluded by a mucous plug.

Pneumonitis — Infection behind a point of partial bronchial obstruction readily occurs. Pneumonitis is seen in about fifteen to twenty per cent of early lung cancers. **Since the signs and symptoms are only those of uncomplicated pneumonitis, and since inflamation readily masks tumor on x-ray, all pneumonitis should be followed by x-ray until it has cleared.**

A history of frequent attacks of pneumonitis calls for investigation of the respiratory tract. The following case history is typical of that given by many patients seen by thoracic surgeons in an advanced stage of pulmonary cancer:

A 53 year old white man was hospitalized with a clinical diagnosis of pneumonia. X-ray revealed a large area of pneumonitis in the right upper lobe. He responded well to medical treatment and was discharged without a follow-up x-ray. He returned six months later, stating that he had never gotten over a residual cough and had had several milder attacks of acute upper respiratory infection. X-ray showed a dense irregular shadow in the region of the former pneumonitis, and enlarged hilar nodes. At bronchoscopy, inoperable carcinoma was found.

Diagnosis:

Chest x-rays, while often not diagnostic, very rarely fail to show an abnormal shadow of some sort while the cancer is still in an operable stage. An infiltrative hilar shadow is most frequent, but shadows cast by atelectasis, bronchiectasis, abscess and pneumonitis secondary to tumor are frequent.

By means of **bronchoscopy,** visualization of the tumor and a positive biopsy are possible in about seventy-five per cent of cases.

Bronchography is useful in revealing the presence of an intrabronchial tumor too small to show on x-ray. Such tumors are usually benign.

When a definite diagnosis cannot be arrived at by other means, in the presence of signs and symptoms suggestive of cancer, exploratory thoractomy should be done routinely. Advising a patient to "wait and see what happens", if the signs and symptoms should be due to cancer, only insures you of the duty of signing his death certificate.

* * * * *

For a Clear Conscience, Be Cancer Conscious!

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LABORATORY AIDS IN THE DIAGNOSIS OF BONE LESIONS

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and
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It cannot be denied that modern medicine is becoming more and more dependent on the clinical laboratory for aids in diagnosis. Even the most astute practitioner who utilizes all of his five senses to aid in diagnosis is not infrequently forced to depend on certain laboratory procedures for help. The clinician practicing in urban areas is more fortunate in this respect than his fellow practitioner in rural areas, who does not have hospital laboratory facilities readily available to him. The diagnosis of diseases producing primary or secondary lesions in the bony skeleton has always presented many difficult problems. While it is true that with the use of a careful history and physical examination and X-rays, a careful clinician can establish the diagnosis of many bone diseases, in certain cases it is possible to arrive at a correct diagnosis only by a correlation of all procurable data, including certain laboratory studies. Laboratory procedures are becoming more and more expensive and there are relatively few patients who can afford an expensive outlay for diagnostic laboratory tests. The physician in a rural area, referring a patient with a bony lesion to an urban hospital, should reasonably expect the period of hospitalization and hospital expense, including laboratory procedures to be kept at the minimum possible. It is the purpose of this paper to so present this subject as to afford an understanding of the interpretation of certain departures from normal laboratory values in the diagnosis of bone lesions.

There are many valuable laboratory tests which are of considerable importance in aiding the clinician to arrive at a correct diagnosis of bone lesions. Valuable as these laboratory aids may be, they are of relatively little value unless they have been preceded by a thorough history and physical examination. With these as a background, the clinician is better able to decide which laboratory examinations to request. The value of any laboratory examination is dependent, primarily, on the reliability and accuracy of the technician performing the respective test. This is particularly true of determinations of blood calcium, phosphorus, and alkaline and acid phosphatase, as these tests are subject to considerable range of laboratory error, and it is suggested that repeat tests be requested in any case in which it is felt that the original examination may have been subjected to laboratory error beyond the control of the technician. The clinical judgment and diagnostic acumen of the physician can save many unnecessary tests and expense to the patient in this respect.

The value of good X-rays in the accurate diagnosis of primary or secondary bone lesions is paramount. In ordering X-rays the roentgenologist should be afforded the benefit of any available clinical or laboratory studies, and he, in turn, is obligated to furnish the clinician with X-ray films of excellent quality and detail.

In considering the differential diagnosis of any bony lesion, the clinician should mentally break the diagnosis down into four broad classifications; (1) Infectious, (2) Metabolic, (3) Neoplastic, (4) Lesions of undetermined etiology. Each of these classifications can then be broken down into the various pathological processes falling within their respective groups. Before considering the various disease processes which produce either primary or secondary bone lesions, it will be well to tabulate the various laboratory examinations which can be of help to the clinician in arriving at a correct diagnosis. As mentioned previously, the ordering of all of such tests in any one case is not only financially prohibitive, but is also an expression of poor clinical judgment on the part of the physician. Generally speaking, the following laboratory examinations will, in the very great majority of cases, furnish the physician with the necessary information to arrive at a correct diagnosis.

(1) *Complete blood count:* To include total red cell count, total white cell count, hemoglobin and a careful differential white blood count, based on the counting of a minimum of one hundred cells seen

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in the stained smear.

(2) *Blood serology for syphilis:* Either a Wassermann, Kahn or a Hinton test.

(3) *Sedimentation rate:* This is normally below fifteen millimeters in sixty minutes for children and below ten millimeters in sixty minutes for adults. It is known that the speed of sedimentation of the erythrocytes is increased in individuals suffering from inflammatory lesions. While there is no uniform agreement as to the cause of this phenomenon, it is believed that the infection present releases products into the blood stream which upset the suspension stability of the erythrocytes in the plasma.

(4) *Sickle cell preparation:* This is of particular value in Southern sections of the United States. Here peculiar sickle shaped erythrocytes are seen in wet smear preparations of patients (usually Negroes or individuals of Mediterranean extraction) suffering from a normocytic, normochromic anemia with an increase in the reticulocytes and basophiles.

(5) *Blood serum calcium:* (Normally a constituent of the blood serum and not of the cells.) Normal values range from nine to eleven milligrams percent in adults and from eleven to 12.5 milligrams percent in children. Parathormone from the parathyroid glands regulates calcium metabolism and maintains a definite level of calcium in the blood. (This is dependent upon an adequate supply of vitamin D.)

(6) *Blood serum phosphorus:* Also normally a constituent of blood serum. Normal values are from 2-4.5 milligrams percent in adults and 4-6 milligrams percent in children.

(7) *Blood serum alkaline phosphatase:* Normal value 1.5-5 Bodansky units in adults. In children, the value will be from 5-12 Bodansky units, depending on the rate of growth of the child. This enzyme, normally found in the blood serum, is increased by osteoblastic activity, and is capable of liberating inorganic phosphorus from organic phosphorus compounds. It is also instrumental in the formation of calcium phosphate, which is deposited in the proteinic bone matrix during the process of bone formation. Alkaline phosphatase is produced by the osteoblasts and the serum content of alkaline phosphatase is an indicator of the activity of these cells. Thus, in lesions where there is rapid bone reproduction, an increase in serum alkaline phosphatase would be expected. This enzyme is excreted in the bile and its serum level is therefore raised in jaundice and liver damage.

(8) *Blood serum acid phosphatase:* This enzyme is produced in the prostate gland, in the male. It has also been reported to be present in females, in minute quantities. The definite origin of acid phosphatase in females is unknown at present. The normal serum content is 0-1.0 Bodansky units. As far as is known, this enzyme only enters the circulation in considerable quantities when a malignancy of the prostate ruptures the capsule of the prostate gland.

(9) *Total blood serum proteins:* This is an expression of the total protein content of the serum, expressed in grams per 100 cc. of serum. Normal values are from 6.0-8.0 grams per 100 cc. of serum.

(10) *Serum albumen-globulin ratio:* The total serum protein is composed of albumen and globulin fractions in the approximate ratio of 2.5-1.5 albumen over globulin. In diseases producing a decrease in the total serum protein, the ratio of albumen to globulin may remain constant, but in still other diseases, with a normal or elevated total serum protein, the relationship of albumen and globulin may be altered, or actually reversed with a greater amount of globulin present than albumen.

(11) *Routine urinalysis:* To include specific gravity, reaction, qualitative albumen and sugar and microscopic analysis for presence of cells or casts.

(12) *Urinary calcium determination:* (Sulkowitz test). To be of value, this test should be determined on a twenty-four hour aliquot specimen. Normally from 0.1 gram to 0.3 gram of calcium is excreted daily. The urinary calcium excreted represents 10% to 30% of total excretion of calcium in normal individuals, the remainder being excreted in the feces. Calcium in the body can be regarded as a threshold subject. If the concentration of calcium in the serum falls below 8 mg. percent, none will be excreted in the urine. When the serum concentration is within normal limits (9-11 mg. percent) and the patient ingests a normal amount of calcium, the urinary excretion should be around 150-200 milligrams daily. On the other hand, when the serum calcium increases above 11.5 mg. percent, the amount of calcium in the urine is always increased.

(13) *Urinary phosphorous determination:* Normally 1-1.5 grams daily. This is usually excreted in the inorganic state. This determination should also

be based on a twenty-four hour aliquot specimen.

(14) *Bence-Jones protein determination:* This is a heavy proteose found in the urine in certain pathological states. This protein precipitates at sixty degrees centigrade and redissolves again at one hundred degrees centigrade. It apparently originates in the bone marrow and its presence in the urine, in states of rapid bone destruction and replacement of bone marrow by pathological cells, is of considerable diagnostic value.

(15) *Bone marrow smear preparation:* Smears of bone marrow obtained from the sternum, ribs, tibia, etc. In questionable bony destructive lesions, examination of such smears by a competent hematologist will frequently furnish invaluable aid in establishing the correct diagnosis.

The above fifteen laboratory examinations, or tests, combined with a good history and physical examination, plus, roentgenograms of good quality will furnish the clinician with diagnostic aids to guide him in making a correct diagnosis. It is not to be implied that even with all the above armamentarium, the correct diagnosis can be made in every instance. Surgical biopsy and histological examination by a good pathologist, of the lesion in question, remains the undisputed criteria for the correct diagnosis. However, this should be held in reserve by the clinician for those questionable cases in which a diagnosis cannot be correctly established by clinical, X-ray and laboratory methods.

Lesions of infectious origin:

1. Ostomyelitis, acute, pyogenic, hematogenous type.
2. Osteomyelitis, subacute or chronic, pyogenic, hematogenous type.
3. Brodie's abscess.
4. Tuberculous osteomyelitis.
5. Syphilitic osteitis.
6. Osteomyelitis (usually localized) of fungicidal or other unusual infectious origin (actinomycosis, blastomycosis, coccidiomycosis, sporotrichosis, etc.)

1. *Osteomyelitis, acute, pyogenic, hematogenous type:* (A detailed history and physical examination are invaluable here.)
   (a) Secondary anemia (may drop as low as 2-3 million R.B.C. in severe cases).
   (b) Increased total white blood count, may be as high as 30-40,000 in the most acute cases in children, with a correspondingly high polymorphonuclear count.
   (c) Increased sedimentation rate: May be as high as 40-50 mm. in sixty minutes in fulminating cases.
   (d) Urine may show some albumen and an occasional toxic granular cast.
   (e) Blood cultures will be positive in cases with active bacteremia or septicemia.
2. *Osteomyelitis, subacute or chronic, pyogenic hematogenous type:*
   (a) Moderate secondary anemia: May be very marked in long standing cases.
   (b) Moderately increased total white blood count (10-18,000) with corresponding increase in polymorphonuclear cells. This will, in a large measure, be dependent upon the chronicity of the lesion and the resistance of the patient to the infecting organism.
   (c) Moderately increased sedimentation rate (15-30 mm. per sixty minutes).
   (d) May have slight albuminuria with an occasional cast.
   (e) If lesion has drained for a long period of time, there may be a decrease in the total serum proteins.
3. *Brodie's abscess:*
   (a) Slight increase in total white blood count with little or no corresponding increase in polymorphonuclears.
   (b) May have slight increase in sedimentation rate, if in an acute exacerbation.
4. *Tuberculous osteomyelitis:*
   (a) Secondary anemia which may be very marked in children.
   (b) Little or no increase in total white blood count (may have moderate leukopenia) but will usually have some increase in lymphocytic count.
   (c) Considerable increase in sedimentation rate, particularly in children. In the acute or active phase of tuberculous osteomyelitis, there may be a gradual increase in the sedimentation rate over a period of days or weeks. The sedimentation rate can be very helpful in determining the activity of the lesion and resistance of the patient to the infection present.
   (d) May have albuminuria with or without

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casts, in an acute fulminating case, or in cases of long chronicity.

5. *Syphilitic osteitis:*
   (a) Positive complement fixation or flocculation tests.
   (b) May have slight leukocytosis with increased ratio of lymphocytes and monocytes in active bony destructive phase.
   (c) Slightly increased alkaline phosphatase in active cases. This may be a very important diagnostic point in the diagnosis of a questionable bony destructive lesion with a positive serology. Apparently the increased alkaline phosphatase is an indication of the attempt of the osteoblasts, at the area of the skeleton involved, to repair the local bony damage being wrought by the infection, but it is also to be remembered that associated liver damage is not infrequently seen in this disease and may also account for some rise in the serum alkaline phosphatase.
6. *Osteomyelitis* (usually localized) of fungicidal or other unusual infectious origin (actinomycosis, blastomycosis, coccidiomycosis, sporotrichosis, etc.). Here the laboratory values will, to a large extent, be expressive of the virulence and the acuteness of the infection present.
   (a) Usually will have a secondary anemia which may be rather marked in an infection of long standing.
   (b) Slight to moderate increase in total leukocytes.
   (c) Increase in eosinophiles in differential count (not always constant).
   (d) Decrease in total serum protein if infection is of long standing and there has been drainage from the granulomatous site.
   (e) Urine may show evidence of long chronic infection by albuminuria and a few scattered granular casts.
   (f) Sedimentation rate usually increased to a moderate extent in these infections.
   (g) Bacteriological studies are of the greatest aid in the diagnosis of these lesions.

Lesions of metabolic origin:

1. Hyperparathyroidism (Von Recklinghausen's disease) (parathyroid adenoma).
2. Juvenile rickets (due to insufficient intake of vitamin D).
3. Renal rickets.
4. Osteomalacia.
5. Osteoporosis.

1. *Hyperparathyroidism (Von Recklinghausen's disease) (parathyroid adenoma).*
   (a) Elevated serum calcium. May go as high as 15-25 milligrams percent.
   (b) Decrease in serum phosphorus. May drop as low as 1-2.5 milligrams percent (this may increase later in this disease if renal impairment develops).
   (c) Increase in urinary excretion of calcium.
   (d) Increase in urinary excretion of phosphorus.
   (e) Increase in serum alkaline phosphatase (if bone lesions are present). It may go as high as 40-50 Bodansky units in very active state of disease.
   (f) Excretion of calcium in stools decreased. In accidental surgical removal of the parathyroid glands, the reverse values are found, namely, drop in serum calcium and elevation of serum phosphorus. If the calcium drops and the phosphorus remains normal, it probably does not mean a loss of parathyroid hormone, but more likely represents blood chemical changes due to steatorrhea.
2. *Juvenile rickets (due to insufficient intake or absorption of vitamin D).*
   (a) Low serum calcium (parathyroid glands cannot metabolize calcium in absence of vitamin D).
   (b) Normal or slightly decreased serum phosphorus.
   (c) Slightly increased serum alkaline phosphatase.
   (d) Absence of calcium in the urine (total excreted calcium is in the stools due to improper metabolism of calcium).
3. *Renal rickets:* Secondary bone changes occurring as a result of altered calcium and phosphorous metabolism due to inability of the kidneys to function properly in chronic nephritis.
   (a) Serum calcium decreased.
   (b) Serum phosphorus increased.
   (c) Increased excretion of calcium in stools.

(d) Decrease or absence of calcium and phosphorus in the urine.
(e) Increase non-protein nitrogen and blood urea.
(f) Secondary anemia.

4. *Osteomalacia:* A bone condition almost rare in this country and seen most commonly in the Far East and Oriental countries; characterized by bony changes secondary to poor dietary intake with poor calcium phosphorus and vitamin D metabolism.
   (a) Decreased serum calcium. Has been known to be as low as five milligrams percent.
   (b) Low to normal serum phosphorus.
   (c) Slightly elevated serum alkaline phosphatase (reparative mechanisms of bone remain normal).
   (d) Decreased excretion of calcium in urine, increased in stool.
   (e) Decreased total serum protein.
   (f) Such cases usually will have anemia and other changes secondary to malnutrition. (In the adult form of this disease, formation of new bone matrix will proceed normally, but there will be little or no calcium deposition in this matrix, due to improper metabolism.) In patients with malignancies of the stomach and intestines, but without metastases to the skeleton, there may be secondary bony changes similar to osteomalacia, due to faulty absorption of food. In such cases the administration of high doses of calcium, phosphorus and vitamin D will be followed by a markedly increased urinary excretion of calcium, whereas in osteomalacia, the urinary calcium will not be increased.

5. *Osteoporosis:* This extremely interesting, yet not well understood, metabolic condition is frequently confused with other secondary bone manifestations; that is, hyperparathyroidism, osteomalacia, multiple myeloma, semi-starvation, immobilization, Cushing's syndrome, etc. In this condition there is actual loss of bony volume and substance, which formerly was believed due to absorption of calcium and phosphorus, but which is now believed to be an actual decrease in bone formation in the presence of constant physiological bone destruction. Here normal bone destruction proceeds as usual, but protein metabolism is decreased with diminution in the amount of bone matrix manufactured and, consequently, a decrease in total bone volume with resultant calcium and phosphorus loss. (That these changes may be in response to adrenal cortical, gonadal or ovarian dysfunction is still under investigation.) The serum calcium and phosphorus and alkaline phosphatase values remain normal; however, a normal alkaline phosphatase value here distinguishes osteoporosis from osteomalacia. There is an increased excretion of calcium in the urine. In osteoporosis of immobilization there is an absence of the normal stress and strain on the skeleton, with resultant less demands on osteoblastic activity. Here, however, there is frequently found an increase serum calcium value in addition to increase of urinary excretion of calcium.

Lesions of undetermined etiology (but probably related to metabolic dysfunction).

1. Osteogenesis imperfecta (fragilitas ossium).
2. Osteopetrosis (marble bones) (Albers-Schonberg disease).
3. Gaucher's disease.
4. Melorheostosis.
5. Hand-Schuller-Christian disease (xanthomatosis) (lipid granulomatosis).
6. Eosinophilic granuloma.

1. *Osteogenesis imperfecta* (fragilitas ossium): A fairly uncommon, congenital disease of hereditary origin, believed to be due to faulty hypoplasia of the mesenchyme. Serum calcium, phosphorus and alkaline phosphatase values are all normal, although there may be elevation of the alkaline phosphatase value, following the frequent fractures associated with this disease.

2. *Osteopetrosis* (Albers-Schonberg's disease), (marble bones). Heredo-familial disease of unknown etiology, characterized by marked increased density of all bones, including the skull. The underlying defect appears to be an inability to resorb bone when new bone is laid down, so that during growth, the old bone is not resorbed.

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(a) Marked anemia—may have erythroblastosis.
(b) Serum calcium and phosphorus normal (occasional case may have slight elevation of serum calcium).
(c) Decreased excretion of calcium and phosphorus in urine.

3. *Gaucher's disease.* This heredo-familial disease of the reticulo-endothelial system produces secondary bone changes, usually most marked in the femoral shaft and femoral neck.
(a) Mild hypochromic anemia.
(b) Leukopenia—differential count remains normal.
(c) Platelet count usually diminished.
(d) Serum calcium, phosphorus and alkaline phosphatase values are all normal.
(e) Bone marrow biopsy, usually shows typical large multinucleated cells of endothelial origin (Gaucher's cells).

4. *Melorheostosis:* A very strange uncommon disease producing hyperostosis and characterized by long, irregular new bone formation simulating the molten wax of a candle and extending along one margin of a long pipe bone. It is a disease of long chronicity and all laboratory values are within essential normal limits. The diagnosis of this lesion should be made by typical X-ray findings alone.

5. *Hand-Schuller-Christian disease.* (xanthomatosis) (lipid granulomatosis). This is another disease of disturbance of the reticulo-endothelial system which produces areas of rarefaction in the membranous bones of the skull, with occasional involvement of other bones. (Letterer-Siwe's disease—another entity of granulomatous origin, with usually fatal outcome before the age of two, is considered by some to be the precursor of Hand-Schuller-Christian disease).
(a) Secondary anemia (due to deposition of lipid material in bone marrow).
(b) Mild leukocytosis in very active cases.
(c) Increased blood cholesterol.
(d) Low specific gravity of urine. (Diabetes insipidus is one of the symptom complexes of this disease.)

6. *Eosinophilic granuloma.* This interesting clinical entity producing granulomatous changes in one or several bones has been reported with increasing frequency recently. The etiology is obscure and all attempts to isolate a specific causative organism have been fruitless. The lesions may resemble a primary skeletal malignancy, multiple myeloma or metastatic bony carcinoma on X-ray findings alone. There are no definite characteristic laboratory changes occurring in this disease. In acute exacerbations of localized lesions there usually occurs a moderately increased sedimentation rate with corresponding slight rise in total white cell count. No increase in circulating eosinophils has been substantiated.

Lesions of neoplastic origin:

1. Osteogenic sarcoma.
2. Chondrosarcoma.
3. Chondromyxosarcoma.
4. Ewing's tumor.
5. Multiple myeloma.
6. Metastatic bone lesions.
7. Paget's disease.
8. Giant cell tumor.
9. Benign bone cysts.
10. Fibrous dysplasia.
11. Osteoid osteoma.
12. Hemangioma of bone.
13. Enchondroma.
14. Osteochondroma.

1. *Osteogenic sarcoma:* The most common primary malignant tumor of bone. Many classical osteogenic sarcomas can frequently be diagnosed by X-ray findings alone, but all too frequently these lesions may be undergoing anaplasia or metaplasia and do not present a typical X-ray appearance. For reasons of simplicity and brevity, the laboratory values usually found in osteogenic sarcoma, chondrosarcoma, chondromyxosarcoma, etc., are listed herewith together, inasmuch as there is very little change in the laboratory values of these closely histologically aligned lesions.
(a) Anemia, almost always present, except in very early phases of these malignancies.
(b) May have leukocytosis with increased polymorphonuclears, if lesion is in rapidly destructive phase.
(c) Serum calcium and phosphorus will usually be within normal limits unless the le-

sion is rapidly destructive, and, in such instances, there will be an increased serum calcium and possibly phosphorus in response to absorption of these minerals from the area of bony destruction.

(d) Serum alkaline phosphatase is usually elevated to a slight or moderate extent in these malignancies. This is an expression of increased osteoblastic activity which usually accompanies these lesions. In general, the degree of the malignancy of the tumor is expressed by the level of the alkaline phosphatase. Of course, in any of these lesions where the bony destruction is all of an osteolytic character with little or no attempt to repair the local damage done, the serum alkaline phosphatase value will not be elevated.

(e) Increased urinary calcium in highly malignant lesions with rapid bone destruction.

4. *Ewing's tumor.* (Endothelial myeloma). Characteristic tumor of children and young adults. Frequently confused with lesions of infectious origin, reticulum cell sarcoma and osteogenic sarcoma. There are no typically reliable laboratory changes occurring in this very malignant lesion, although anemia is rather constant as the disease progresses, and in the rapidly destructive phase of Ewing's tumor there may be leukocytosis with some increase in the serum alkaline phosphatase. (The diagnosis of Ewing's tumor, even with surgical biopsy, can frequently be in doubt, as even the most experienced pathologists debate as to the histological differentiation between Ewing's tumor, reticulum cell sarcoma and metastatic neuroblastoma.)

5. *Multiple myeloma:* Primary bone tumor arising from myeloid tissue of marrow. Frequently confused with osteoporosis, hyperparathyroidism and metastatic bone lesions.

(a) Marked secondary anemia (2-3.5 million RBC).

(b) Approximately 25% of proven cases have slight leukocytosis.

(c) Differential count will show some increase in mononuclear elements with circulating myelocytes, ranging between 1%-10%. Some reports of finding circulating plasma cells are on record, but their occurrence is very unusual.

(d) Albuminuria. May be very heavy in late stages.

(e) Bence-Jones proteinuria occurs in 50-65% of cases (a negative Bence-Jones protein determination does not rule out multiple myeloma, and an occasional false positive can occur, but a positive Bence-Jones proteinuria is strong evidence in favor of a diagnosis of multiple myeloma). The test should be repeated frequently, as transitory Bence-Jones proteinuria is the rule, rather than the exception, in multiple myeloma.

(f) Usually will have increased total serum protein—7.5 to 8.5 grams. Reversal of the albumen and globulin ratio is not uncommon and is of considerable differential diagnostic value, if present.

(g) Serum calcium, phosphorus and alkaline phosphatase values are usually normal, although an occasional case may have a slightly increased alkaline phosphatase and if there is severe renal impairment, the serum inorganic phosphorus will be elevated as an expression of retention of phosphorus by the damaged kidneys.

(h) Bone marrow puncture and smears reveal typical infiltration by large numbers of plasma cells.

6. *Metastatic bone lesions:* Systemic malignancies with metastasis to the bony skeleton present very difficult differential diagnostic problems. The importance of a good history and a careful physical examination cannot be over-emphasized in these cases. Such bony metastases will be either osteolytic or osteoplastic in nature and, if the involved bone attempts to repair the damage brought about by the metastatic cells, the serum alkaline phosphatase will respond accordingly. Metastases from the prostate will usually provoke osteoblastic activity with a corresponding rise in serum alkaline phosphatase values, plus a rather marked rise in serum acid phosphatase value. On the other hand, metastatic lesions from the kidney, breast, thyroid, lung, etc., are usually osteolytic and show no increase

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in the alkaline phosphatase level. In such osteolytic bone lesions with absorption of calcium and phosphorus from the area of destruction, there will result slight elevations of the serum calcium and phosphorus levels. Correspondingly will be found an increased excretion of these minerals in the urine. Even in some osteolytic metastatic lesions there will be some increase of serum alkaline phosphatase, but, in general, the higher the serum alkaline phosphatase value, the more normal are the serum calcium and phosphorus values. It is to be remembered also that with metastatic bone lesions present there very well may be liver metastasis also present and an elevated serum alkaline phosphatase may be indicative of liver damage present. Not every case of metastatic prostatic carcinoma will show an elevated serum acid phosphatase value, but the test should be repeated at intervals, as the great majority of such cases will present an elevated acid phosphatase value sooner or later in the course of the disease. An osteolytic type of bony metastasis may resemble the lesion of multiple myeloma on X-ray and, in such instances, carefully chosen and accurately run laboratory procedures will greatly aid the clinician in distinguishing the two lesions.

7. *Paget's disease* (osteitis deformans): In well developed cases of this very interesting condition with involvement of several bones, the X-ray appearance will be rather characteristic, but in early lesions of Paget's disease or with monostotic involvement, the X-rays will not be entirely characteristic and frequently the lesions of Paget's disease will closely resemble metastatic prostatic carcinoma. The values of serum calcium and phosphorus are within normal limits in the great majority of cases. The alkaline phosphatase value, on the other hand, is almost always elevated in Paget's disease and may go as high as 150-200 Bodansky units in some cases, although values of 20-50 Bodansky units are more characteristic. In Paget's disease, with a pathological fracture followed by immobilization of the fracture, there may occur rather marked demineralization with resultant rise in serum calcium and phosphorus values, with increased excretion of calcium in the urine, but these values are uncommon. In general, the level of serum alkaline phosphatase will remain rather constant in Paget's disease over a period of months or years. In lesions presenting X-ray characteristics of both Paget's disease and metastatic prostatic carcinoma, the presence of an elevated serum acid phosphatase will be helpful in establishing the correct diagnosis. In general, other blood chemistries values and urinary findings are within normal limits in Paget's disease.

The following group of bony lesions present no characteristic laboratory findings. Surgical biopsy and histological examination should be employed to establish the correct diagnosis if the X-ray appearance of the lesions is not entirely characteristic.

8. *Giant cell tumor* (osteoclastoma).
9. *Benign bone cysts.*
10. *Fibrous dysplasia of bone.*
11. *Osteoid osteoma.*
12. *Hemangioma of bone.*
13. *Enchondroma.*
14. *Osteochondroma.*

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401 Medical Arts Building

CLINICOPATHOLOGICAL REPORTS

From the Case Records of the Medical College of Virginia and the University of Virginia Hospitals

HARRY WALKER, M.D., *Editor*

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CASE NO.: 74

A 37 year old colored female was admitted to St. Philip Hospital because of fever and aching "all over". Approximately two weeks before admission she developed a sore throat with cough and fever and generalized aching. The sore throat and cough subsided in a few days, but the fever persisted and the aching became more and more severe to the point where the patient was "sore all over". The temperature apparently was sustained though it seemed to be lower in the evenings. She had no chills, no rash, and there had been no change in bowel habits. She had vomited twice.

The patient had been a known hypertensive since a previous hospital admission three years earlier. At that time she was admitted because of suspected pregnancy. Her blood pressure was recorded at 238/148. Her general physical examination was negative except for a mass in the region of the left salpinx and her laboratory studies, including blood count, urinalysis, NPN, Mosenthal and serum proteins, were all normal. A diagnosis of hydrosalpinx was made but the patient refused surgery and was discharged. Some months after this admission she apparently developed congestive failure and the referring doctor indicated that she had been maintained on digitoxin, mercurials and barbiturates. In spite of this treatment she continued to have attacks of pulmonary edema from time to time. She also complained of frequent headaches. She had, however, been getting along fairly well for sometime prior to the onset of the present illness.

She had had five pregnancies in thirteen years, the last five years before this final admission. All were home deliveries and were described as normal.

The patient lived in the country and drank unpasteurized milk. She stated that there were many rats around her home.

Physical Examination: Temperature 101.6. Pulse 130. Respiration 40. Blood pressure 190/120. The patient was well developed, moderately obese, appeared acutely ill and complained of generalized soreness and aching. There was moderate retinal arteriosclerosis with some A-V nicking, but no hemorrhages, exudates or papilledema. The neck veins were slightly distended. The lungs were clear. The heart appeared enlarged with a diffuse PMI in the 5th left intercostal space, 12 cms. to the left of the midclavicular line. There was a gallop rhythm. Also, there was a systolic thrill and a harsh systolic murmur in the 2nd and 3rd interspaces to the left of the sternum and a softer systolic murmur at the apex. The liver was palpable 2 fingers breadth below the right costal margin and was non-tender. The abdomen was otherwise negative as were the pelvic and rectal examinations. There was no edema of the extremities and the pedal pulses were easily felt. No skin eruption was noted but the skin was described as "tight and brawny with generalized tenderness". The joints did not appear involved. There was no significant lymphadenopathy.

Laboratory Data: Urine acid, 1.015 specific gravity, 2+ albumin, negative sugar and acetone, occasional WBC and a few granular casts microscopically. RBC 4,200,000, hbg. 12.5 grams, WBC 21,200 with 98 polys., 1 bas., 4 lymphs., and 4 monos. NPN 41 mgm.%. Sedimentation rate 60 mm. in one hour. Chest x-ray showed an enlarged aortic type heart with a 69% C-T ration and diffuse widening of the aorta. The lung fields were clear. An EKG showed sinus rhythm with a rate of 125 per minute, the P-R interval 0.18 and the Q-S interval 0.08. The above changes were interpreted as left ventricular strain with digitalis effect.

Following admission the patient's temperature remained around 102 (R) for the first five days. The pulse also remained elevated, 100-110/min. Several blood cultures were obtained, all of which were subsequently reported as negative, and the patient was started on crysticillin, 600,000 units every 6 hours, and streptomycin, 0.5 grams every 6 hours. She was continued on digitoxin and given codeine for pain. She appeared to improve somewhat after the first week and her temperature became almost normal. However, she continued to complain of generalized soreness and aching and frequently complained of

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pains in her chest posteriorly. A repeat WBC shortly after admission showed 19,800 cells with 79 polys., 5 eos., 1 bas., 12 lymphs., 3 monos. and several repeated counts showed a persistent leukocytosis to as high as 26,000 with a differential similar to the second count. The hemoglobin dropped slowly to around 9 grams. Repeated urinalyses showed no change until the day of death when 10-12 RBC were noted for the first time. The NPN rose gradually to 184 mgm.%.

Repeated chest films showed several patchy densities above the dome of the right diaphragm and, later, a density in the left costo-phrenic angle.

Following the apparent initial improvement the patient started going downhill, became more and more lethargic, and died on the 27th hospital day. An autopsy was done.

DISCUSSION BY DR. WILLIAM B. PORTER.*

Essentially this case is a Negro woman, 37 years of age, who was admitted to St. Philip Hospital apparently acutely ill. Her physical complaints consisted of fever, which followed what was thought to be an upper respiratory infection. The illness progressed, the fever continued, and she complained of soreness all over her body. There were no chills or rash, and there had been no change in bowel habits. She had vomited twice. The patient was a known hypertensive individual, having been in the hospital three years previously with a blood pressure of 238/148, and I presume the discharge diagnosis was essential hypertension. It is interesting that she apparently had a mass in her abdomen and the surgeons were quite anxious to remove it. It was not removed and apparently the mass disappeared spontaneously. This sounds like the patient's judgment was better than that of the surgeons. Another interesting point is that this patient had apparently been kept in contact with, presumably through the out-patient clinic, because the protocol gives us the history of recurring attacks of what was described as pulmonary edema. She also complained of frequent headaches but the final rather paradoxical statement is made that though she had been having recurring attacks of pulmonary edema, she had apparently been getting along quite well prior to admission. This is very interesting and makes one wonder whether the patient was having true pulmonary edema or whether she was having some respiratory disturbance of an asthmatic nature.

The patient had been pregnant five times in 15 years. Her deliveries were at home and from the history nothing unusual happened.

The next statement, which to my mind is probably a red herring, is that the patient drank unpasteurized milk and lived in an atmosphere of many rats.

We have then a patient with hypertension (blood pressure 190/120), probable cardiac insufficiency, fever, tachycardia, and an accelerated respiratory rate. Then we come to what I believe are two significant findings: The first is the absence of any gross changes in the optic fundi and secondly, the presence of diffuse, wide-spread soreness over her extremities. Along with those findings, we note that she has an enlarged heart and a gallop rhythm. These too, I believe, are significant findings. She had murmurs, systolic in time, which one would have great difficulty in evaluating for the obvious reason that a febrile patient, with an enlarged heart, might have murmurs under the conditions existing in this individual, both from a probably dilated as well as hypertrophied heart. There another statement made which I think may also be of considerable importance; that is, the patient's skin was tight and brawny with generalized tenderness. A tight and brawny skin with generalized tenderness would certainly seem significant.

We now come to the laboratory data. I am impressed with some of these findings. In the first place, the urine is abnormal, though the abnormalities are not too specific—low specific gravity and some casts, but with no red cells or white cells. There was no definite anemia at that time, but there was a definite leukocytosis with a very rapid sedimentation rate. I have before me the electrocardiogram on this patient. It indicates to me the findings which one would expect in an individual with an enlarged heart and changes in the heart muscle probably secondary to coronary disease. Perhaps some of the experts would like to look at this tracing. I would not feel that the digitalis effect was too evident in this particular tracing. The changes in the T waves are more suggestive of changes in the heart muscle.

I have glanced at the x-ray films but I would like very much for Dr. Mandeville to discuss them.

*Professor of Medicine, Medical College of Virginia, Richmond.

DR. MANDEVILLE*—These four films which we have here suggest to me an enormous heart, a very large heart, and I believe there is more enlargement of the left than the right heart. I think there is enlargement of the thoracic aorta. There are a few patchy densities which could be infarcts. I do not see the widespread patchy densities sometimes seen in periarteritis nodosa. I do not think there is anything typical about periarteritis nodosa and from an x-ray standpoint would not have gone overboard for such a diagnosis. There could be congenital heart lesions in the film but there is nothing typical. We have no barium swallow to tell us whether the left auricle is large. In other words, if there are any atrial or ventricular defects, I cannot find evidence of them on these films. So I would have to be satisfied with an enlarged heart, possibly with an aortic lesion, congestion, and, from a pure roentgen standpoint, pulmonary infarcts.

DR. PORTER—It is the type of x-ray which one would expect in any patient with chronic hypertensive disease who had reached the stage of failure.

Now for a brief comment on therapy in this individual. She was treated with antibiotics probably because of the uncertainty as to the diagnosis. She was continued on digitalis, and certain remedies were used for the relief of pain. The patient complained of pain in the posterior chest. I do not know just what that means, but it may be significant. Another interesting thing which may not mean too much, but I think is significant, that her leukocyte count continues to be elevated to as high as 26,000 per cu. mm. Also, there was an increase in the polymorphonuclear count and the appearance in the blood smear of as many as 5% eosinophiles. The presence of 5% eosinophiles with a white count of 19,800 leukocytes would give a total eosinophile count of about 1000 per cu. mm. which is a gross eosinophilia. I think sometimes we are inclined to ignore the presence of eosinophiles because of a small percentage when actually it is important to have an actual eosinophile count. We have in this patient, then, a leukocytosis with an absolute eosinophilia. That may mean something or it may not. It is to be remembered, however, that in patients who have a polymorphonuclear leukocytosis, the presence of an absolute eosinophilia indicates that you are dealing with a rather exceptional situation. The disappearance of the eosinophiles from the peripheral blood in polymorphonuclear leukocytosis is the rule. It is an exception to find an absolute eosinophilia.

After several days this patient gets what seems to be a moderate remission but then reverts rapidly back to her former state with continued fever, soreness and aching. In summary then, we are dealing with a patient who is a known hypertensive who develops a febrile illness, and in whom there is a rapid downward course, particularly as concerns the kidneys and cardiac muscle and this is accompanied by the local phenomenon of either myositis, neuritis, arteritis or perhaps all three, a leukocytosis, an eosinophilia—again all of this occurring in a patient who has known hypertensive vascular disease.

Briefly, I would only like to mention what I imagine were the diseases thought of when this patient entered the hospital. Due to her peripheral symptoms, I am perfectly certain that the resident staff, and whoever else saw the patient thought of Weil's disease. The patient's intimate association with rats would also bring this disease to mind. The things against Weil's disease are the degree of leukocytosis and the late development of renal failure without any evidence of jaundice or hepatic failure. I think it is sometimes logical for one to make a diagnosis of Weil's disease without evidence of hepatic failure, but Weil's disease attacks the kidney and liver at the very outset of the disease. It is not something that the patient develops later in the course of her illness. I, myself, would therefore discard Weil's disease as a possibility.

The next disease which was obviously thought of is trichinosis. I am certain that someone thought of this to explain what had happened to the patient to account for the acute symptoms she presented. She was already known to have hypertensive disease. Trichinosis does not kill one that way. Almost invariably, and I would say always, severe trichinosis is accompanied by gastro-intestinal symptoms at the onset of the disease and, in addition to that, those patients who have trichinosis develop a leukocytosis of 25 to 30 thousand, maybe, but they do not have 5% eosinophilia, they have 15 to 20% eosinophilia. I merely mention trichinosis as a disease which may produce fever and muscular pain, but I do not think this patient had this disease.

The next disease, which was certainly thought of

*Dr. Frederick B. Mandeville, Professor of Radiology, Medical College of Virginia, Richmond.

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in this patient is so-called dermatomyositis. Dermatomyositis is a strange disease; an inflammatory disease involving the skin and the muscles, but it is rarely so rapidly fatal. There is marked thickening of the skin sometimes with a dermatitis, and marked tenderness of the muscles. Leukocytosis, if present is mild. The disease does not produce any particular change in either the heart or the kidney. Scleredema was probably thought of but scleredema does not act this way clinically. Scleredema is an afebrile disease running a very long and progressive course. It is characterized by both gastro-intestinal and cardiac complications usually of a very serious order, but I do not recall ever seeing a patient with scleredema who acted in the same way as this patient did.

I think one should think about those conditions which may occur in a patient with vascular disease which makes that vascular disease prove fatal in a very rapid manner. The first one of these things that most of us here would think of would be the so-called malignant phase of essential hypertension. It is one thing that can kill patients with vascular disease rapidly. However, there are so many things in this patient against that, that would not be explained by such a diagnosis. For example, I have never seen a patient with malignant hypertension who died in renal failure who did not show gross changes in the optic fundi. The second thing is that it is an afebrile disease. The third point is that the patients ordinarily do not have any peripheral symptoms. I think, therefore, that this condition is out of the question because it will not explain anything except death, and renal failure.

Another cause for rapid decadence in patients with vascular disease is the development of pyelonephritis. We have been more and more impressed with the importance of pyelonephritis as an intercurrent factor producing a rapid decline in patients with essential hypertension. Pyelonephritis is, of course, a possibility, but I cannot imagine making that diagnosis in the absence of urinary changes which would suggest that possibility. There was no pus in the urine and only in the last specimen were there any red cells. The diagnosis of pyelonephritis in a febrile patient with a leukocytosis of 25 to 30 thousand without pus, almost gross pus, in the urine would be almost an absurdity. Pyelonephritis is, however, one of the causes of a rapid exodus in patients with essential hypertension.

I always like to go about discussing a case as it is recommended in a book I have on psychoneurosis. The author has a paragraph in this book at the beginning of a chapter on psychoneurosis on the diagnosis of psychoneurosis. He states that a diagnosis of psychoneurosis by a process of elimination is fundamentally unsound and is usually totally incomplete. The diagnosis of a psychoneurosis should be made by frontal attack for several obvious reasons. In the first place, if you make the diagnosis of psychoneurosis by frontal attack, you do so because you recognize the sum total of this individual's problems, and size the patient up as having that sort of a malady. This is so important because there is no reason why psychoneurosis may not have an associated disease or condition which may have nothing to do with his presenting symptoms. When you go about such a diagnosis by the process of elimination, you have missed the main issues.

In this particular patient then, I would rather approach the diagnosis from this point on as a direct frontal attack. I will do so by trying to decide whether there is any one disease which will account for all of the symptoms and signs which this patient had. In the first place, we have a patient with a febrile disease characterized by rapid decadence of the heart muscle and the kidneys and probably at the same time with a peripheral lesion of arteritis, neuritis or myositis. We also have an individual who has hypertension. Now, it seems to me that when you are confronted with a patient of this kind who in addition to these things has a leukocytosis and an absolute eosinophilia, that anyone who did not consider the diagnosis of pariarteritis nodosa would be greatly amiss. Periarteritis nodosa can explain everything in this patient. That may not be what the patient had, but unless it is periarteritis nodosa, it will be very difficult to explain many of the symptoms and signs which this patient had.

What is periarteritis nodosa? I am thinking now about the clinical disease as described by Kussmaul and Maier in 1883. There are many diseases which have periarteritis or panarteritis, but I am beginning to get the idea in my head that in many instances the pathologists should not make an unequivocal diagnosis of periarteritis nodosa without knowing something of the clinical history of the patient. What we are trying to describe is a disease which has the

following clinical characteristics:

1. It is a disease which has the clinical course of an infectious process, fever, leukocytosis as a rule, and other symptoms of an infectious process.
2. It is a disease which has regional symptoms depending on the degree of involvement of the various vascular structures supplying these areas.
3. At sometime or another in the course of this disease, there is a leukocytosis and an eosinophilia. This condition may be present at one stage of the disease, and disappear entirely within a week or ten days later. I have followed one patient with what I believed to be periarteritis nodosa for a period of 2 years. That man has had a leukocyte count varying from 30 to 50 thousand with an eosinophile count from 1% to as high as 22%. One reaches a conclusion that if a patient with periarteritis nodosa is followed sufficiently long, they will all have an eosinophilia at one time or another.

I will not take up your time to go into a lot of side issues. I would simply feel that we are dealing with a patient who three years ago had what was believed to be essential hypertension and who now developed the syndrome known as periarteritis nodosa. I think that she died from renal failure and cardiac failure.

HOSPITAL DIAGNOSIS: Congenital heart disease (? pulmonic stenosis). Subacute bacterial endocarditis.

DR. PORTER'S DIAGNOSIS: 1. Periarteritis nodosa.
2. Essential hypertension.

PATHOLOGICAL REPORT: Dr. G. R. Hennigar*.

The heart weighed 600 grams. Both ventricles were obviously hypertrophied, and in addition, there was considerable dilatation of the right side of the heart. The width of the left ventricular myocardium, at the mid-point between the base and apex was 2.3 cms. (normal 1.4-1.6 cms). The width of the right ventricular myocardium at the mid-point between the base and apex was 1 cm. (normal .4-.6 cms.). The right atrium except for some dilatation was not altered. An interesting finding was the presence of a small medial cusp of the tricuspid valve. The tip of the cusp was firmly adherent to the interventricular septum. The chordae tendineae were short and fine (fig. 1).

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Fig. 1—The right side of the heart is opened. The tip of the pointer shows the tiny chordae and valve cusp adherent to the endocardium of the interventricular septum. The remainder of the chordae are thickened and shortened.

The remainder of the chordae are short and thick. Whether these abnormalities represent a congenital malformation such as that resulting from fetal endocarditis or whether they are manifestations of old rheumatic endocarditis is not clear. Other stigmata of healed rheumatic affection were present in the heart. These comprised periarterial fibrosis around the vessels of the myocardium, subendocardial fibrotic plaques (rheumatic arteritis), and small nodules on the cusps of the aortic and mitral valves. This latter group of morphological rheumatic manifestations, we do not feel were marked enough to cause impairment of cardiac function sufficiently to be clinically significant. The rheumatic arteritis is shown in fig. 2.

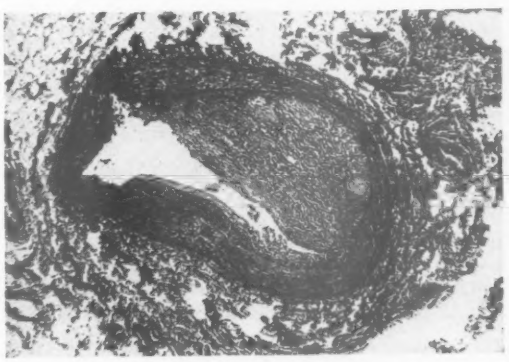

Fig. 2—Rheumatic coronary arteritis. Subintimal foci of connective tissue greatly narrow the lumen of the vessel, Verhoff-van Gieson.

At autopsy, small nodules or "beads" were seen along the larger branches of the coronary arteries, especially the anterior branches of the left (fig. 3).

Fig. 3—The arrow points to one of the nodules on a branch of the left coronary artery.

Microscopically, the wall of the vessel and adjacent areolar tissue of the epicardium were converted into eccentric nodules of connective tissue. Elastic tissue stains showed fragmentation of the elastic fibers with herniation of the wall of the vessel outwards. No fresh collagen necrosis of the vessel wall was appreciated.

In contrast to this "healed" lesion fresh or recent necrotic vessels were found in the diaphragm. Surrounding these was a rich cellular infiltrate consisting of eosinophil and neutrophilic granulocytes, lymphocytes and plasma cells (fig. 4). In addition, in the diaphragm, vessels which showed "fresh" and "healed" lesions combined, in the same vessel, were evidenced (fig. 5). The initial necrosis of the wall leads to weakening and aneurysmal dilatations occur. Later these become filled in with fibroblasts (see left hand side of vessel in fig. 5). In this mass of connective tissue granules of hemosiderin and bilirubin are seen; evidence of hemorrhage probably at the time the aneurysm "leaked" or ruptured.

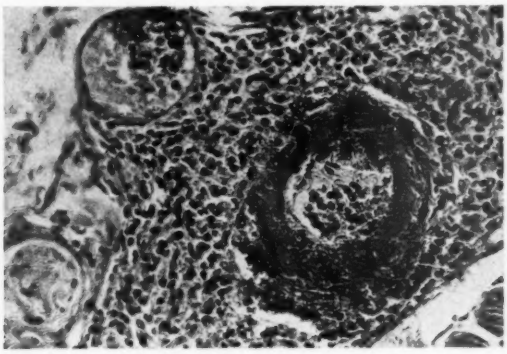

Fig. 4—Vessel of diaphragm. Characteristic necrotizing lesion (collagen necrosis, "fibrinoid" necrosis) seen in acute periarteritis nodosa. The wall is an acidophilic smudge. Granulocytes, as well as lymphocytes are conspicuous in the cellular infiltrate.

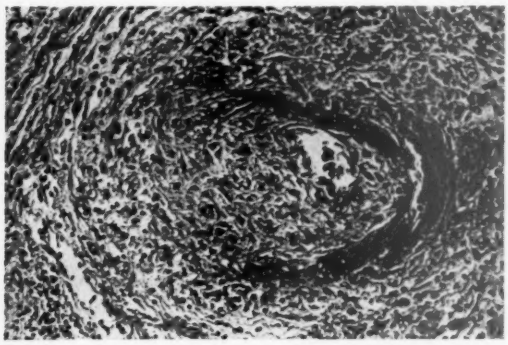

Fig. 5—The wall of the vessel is completely replaced by fibroblasts with narrowing of the lumen. The dark areas in the wall to the right represent recent necrosis.

The diagnosis of periarteritis nodosa was made by

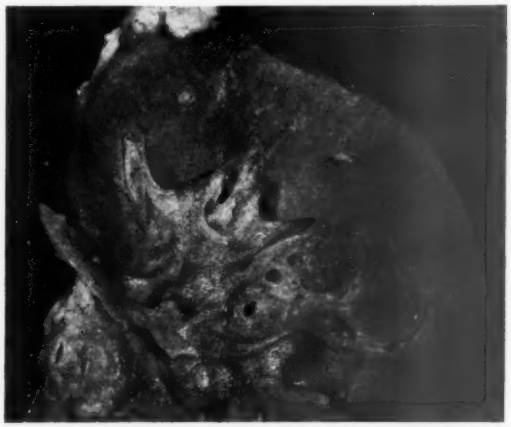

Fig. 6—The vessels at the bases and tips of the pyramids are surrounded by white, translucent "sheaths" of connective tissue.

the prosector, Dr. Jacqueline Kirk on the basis of thick, white, translucent sheaths surrounding the vessels of the kidney, liver and adrenal (figs. 6, 7, 8).

Fig. 7—Note similar "sheaths", perivascular in location in the liver.

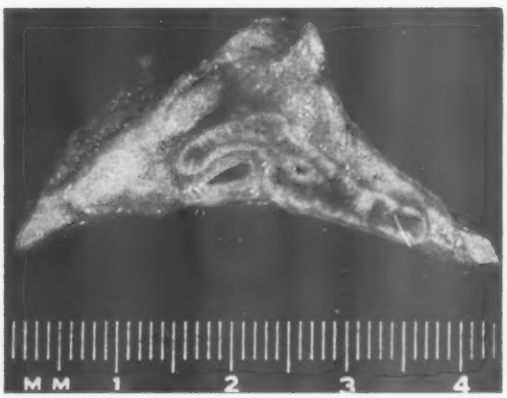

Fig. 8—Identical "sheaths" surround the vessels of the adrenal medulla. The periadrenal region is composed of confluent white fibrous tissue.

The microscopic findings in the adrenal consisted of pseudo-tubular change in the zona fasciculata, fragmentation of the reticulum supportive framework, loss of lipoid throughout the cortex, irregular amount of pigment in the zona reticularis. We do not feel that we are able to interpret these microscopic findings other than to say that they are the morphological findings associated these days with adrenal exhaustion as a result of a variety of stressor agents. In hypertension and periarteritis the vessels in the periadrenal fatty connective tissue are constantly and severely involved so that the adrenal parenchymal effects may, at least in part, depend on these vascular changes, rather than directly as a result of the hypertension or collagen disease on the cells, as some investigators have suggested. Certainly, the profound vascular changes would be expected to lead to some degree of hypoxia to the underlying adrenal cortex.

By the same token the kidneys, involved in 80-95% of cases of periarteritis nodosa have no constant gross or microscopic characteristics, for the arteriolosclerosis and arteriolonecrosis of hypertension in far the majority of cases distort the picture.

The pancreas was interesting in that the islets in sections from the body of that organ were very large. This finding may be indicative of some degree of adrenal insufficiency.

We feel that the patient probably died from the effects of the periarteritis and hypertension, more particularly, left ventricular failure and some degree of renal insufficiency.

An understanding of the etiology (or etiologies) and pathogenesis of the clinical group of diseases which selectively insult collagen, is to say the least obscure. That there is a common morphological denominator, namely, collagen ("fibrinoid") necrosis and that there are distinct clinical entities (lupus erythematosus, periarteritis nodosa, rheumatic diseases, etc.) with of course some overlapping, is not disputed. Thus, it would seem that the bulk of "the riddle of the collagen diseases" will have to be solved by other than the morphologists.

PATHOLOGICAL DIAGNOSES:

1. Periarteritis nodosa, generalized.
2. Left and right hypertrophy and dilatation ventricular myocardium.
3. Adherent medial cusp of tricuspid value to endocardium of the interventricular septum (?congenital, ?rheumatic).
4. Generalized arteriolosclerosis.
5. Arteriolosclerotic nephritis.
6. Healed rheumatic pancarditis.
7. Eosinophilia bone marrow and spleen.
8. Hyperplasia islets of Langerhans.

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MEDICAL SOCIETY OF VIRGINIA

Report of Delegates to A.M.A.

The 1951 regular session of the House of Delegates of the American Medical Association was held at the Hotel Traymore, Atlantic City, New Jersey, June 11-15, 1951. For the first time in its history the House included two medical students, president and vice-president, respectively, of the recently organized Student American Medical Organization.

Several resolutions were introduced looking toward creating one class of membership and abolishing the present distinction between members and fellows. No final action was taken, but the matter was referred to the standing committee of the House on Constitution and By-laws, and a report with recommendations may be expected at the interim session of the House in December. Dues for 1952 were set at $25.00 which includes subscription to the Journal.

The very important subject of inspection and accreditation of hospitals concerning which there are several conflicting views again came up for consideration. The previous proposal that a joint commission composed of representatives from the A.M.A., the American College of Surgeons, the American College of Physicians, and the American Hospital Association be formed for that function was adopted. It is expected that this plan will be approved in the near future by the several groups interested.

As reports of officers and trustees indicated that the A.M.A.'s publicity campaign had been successful, it was decided to slow down on expenditure in this direction. However, Whitaker and Baxter will be retained in a consulting capacity. A plan proposed by the Board of Trustees to appoint a committee of prominent laymen to advise the board, particularly as regards the viewpoint of the public in matters pertaining to medical care, was approved.

Other matters considered included health insurance, tax relief for physicians' expense for postgraduate study, relief for medical schools distribution of physicians and distribution of interns. As regards the distribution of interns, an advisory committee to the Council on Medical Education was approved to study the problem and make recommendations.

The House was again addressed by a prominent labor leader, this time Mr. Dave Beck of Seattle, Washington, Executive vice-president of the International Brotherhood of Teamsters. His address entitled, "Government Medicine—Danger Ahead", was broadcast and also printed in the A.M.A. Journal for June 30th. He expressed very forcefully his support of the efforts of the A.M.A. and his opposition to government control of medical care.

Dr. John Cline was installed as president. Dr. Louis H. Bauer of Hempstead, New York, was chosen as president-elect, and Dr. Oscar B. Hunter of Washington, D. C., as vice-president. Dr. David Allman of Atlantic City was elected to succeed Dr. Bauer as trustee and Dr. Walter B. Martin was reelected to the Board for another term. The 1951 Clinical Session and interim meeting of the House of Delegates will be held in Los Angeles December 4-7, 1951.

J. M. EMMETT
H. B. MULHOLLAND
J. M. HUTCHESON
Delegates

MENTAL HEALTH

JOSEPH E. BARRETT, M.D.,
Commissioner, Department of Mental Hygiene and Hospitals.

What Happens to Mental Hospital Patients?*

What is the probability of a patient leaving a mental hospital after admission? This question is often asked. Another question follows: How long do patients remain in the hospital after admission? These questions have been of such importance to the Department of Mental Hygiene and Hospitals and to families or friends of patients that an analysis has been made to show the status of patients (in or out of hospital) six months and one year after their admission dates.

This study indicates that the probability of a patient being released from a mental hospital is greatly reduced after a stay of longer than six months. An analysis of the admissions of mentally ill persons for the year ending June 30, 1950, shows the following: For those admitted as first admissions 40.2% were discharged or released on trial visit during the first six months. By the end of a year, from date of admission, 46.1% were discharged or released on trial visit. This was a difference of only 5.9% between six months and one year of hospitalization. Those remaining in hospital were 42.3% at the end of six months, and 31.0% at the end of of a year from date of admission. The death rate was 17.5% for six months and 22.9% for one year. Chart Number 1 shows this graphically.

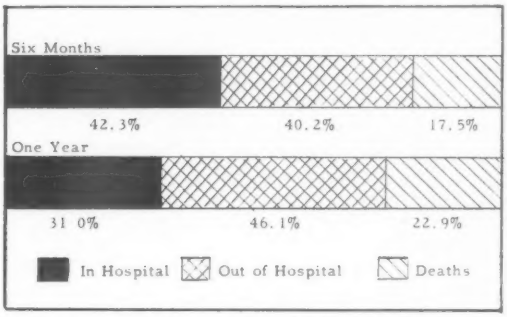

Chart 1. Comparison of Status of First Admissions Six Months and One Year from Date of Admission.

The picture in regard to readmissions because of mental illness is somewhat different. Readmissions constitute 35% of all admissions. At the end of 6 months 51.4% of the readmissions have been released, 7.8% have died. It is noted that the death rate of readmissions is much smaller than the first admissions. This is because the readmission rate among the old age group is much smaller, in fact, practically non-existent. Chart Number 2 shows this picture graphically.

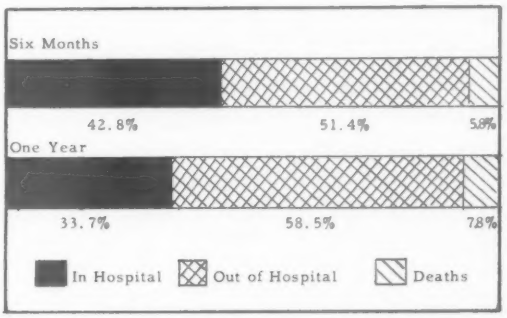

Chart 2. Comparison of Status of Readmissions Six Months and One Year from Date of Admission.

Now if we compare the first and readmissions in a study of all admissions, we have the following: At the end of six months 44.0% of all admissions have been released; 13.6% have died. At the end of one year 50.4% of all admissions have been released; 17.7% have died. Chart Number 3 shows this graphically.

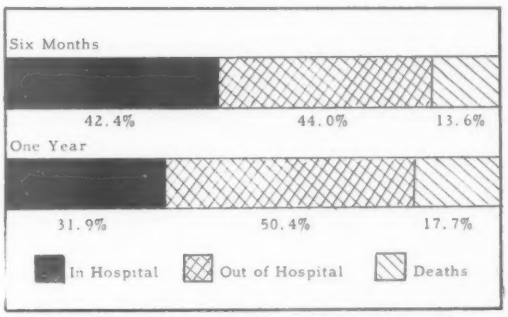

Chart 3 Comparison of Status of All Admissions Six Months and One Year from Date of Admission.

From these studies it may be seen that patients who are not sufficiently recovered or improved within six months following their admission have a rela-

*Prepared by Edna M. Lantz, Statistician, Department of Mental Hygiene and Hospitals.

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tively smaller probability of leaving the hospital. Therefore, intensive treatment of patients during the first months following admissions appears to produce better results than a longer term of hospitalization.

In a statistical review of individual hospitals in this particular analysis it was noted that those hospitals that had a higher ratio of professional personnel to patients also showed a higher percentage of discharges and trial visits during the first six months following admission.

PUBLIC HEALTH

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

Diphtheria

The following table lists the incidence and fatality from Diphtheria as reported in Virginia since 1920:

DIPHTHERIA IN VIRGINIA, 1920-1951

Year	No. Cases Reported	Rate (Cases per 100,000 Population)	Number Deaths	Fatality Rate
1920	4370	188.79	334	7.6
1930	2210	91.3	172	7.8
1940	709	26.4	51	7.2
1941	754	27.3	44	5.8
1942	699	24.9	42	6.0
1943	395	13.8	25	6.3
1944	329	11.8	23	7.0
1945	577	20.5	42	7.3
1946	548	18.3	43	7.8
1947	273	8.9	23	8.4
1948	265	8.4	13	4.9
1949	220	7.0	12	5.5
1950	192	5.8	11	5.7
1951*	88	2.6	5	5.7

*(Through September 30)
Figures for 1951 are provisional.

Although there has been an encouraging and steady decline (except in 1945-46) in the incidence of Diphtheria in Virginia, we should not be complacent, recognizing that the Virginia case rate for 1950 (5.8) is higher than for the United States as a whole (3.9). The higher incidence in 1945-46 seemed to confirm the apprehension that there would be an increase in diphtheria in the United States, reflecting the high attack rate in Europe. During the winter of 1945-46 there was a high incidence of diphtheria in Europe and the case fatality rate was also high. It was felt, therefore, that the exposure of thousands of American soldiers to these organisms of apparently high virulence, made possible the return of many diphtheria carriers. Fortunately this was not the case.

However, the progressive decline in incidence should not give a sense of false security. In control of diphtheria, eternal vigilance is still the price of security. Such vigilance demands widespread immunization and the detection of carriers of virulent organisms. It is recommended that immunization against diphtheria (preferably in conjunction with pertussis and tetanus) be given as early as the 3rd month, and many physicians recommend such immunization as early as the second month of life. Second and third injections are given at intervals of 1 month and a booster dose is recommended at 18 months and before entering school.

The handling of the carrier is often a difficult problem. Some success has been reported from the intensive use of penicillin during the course of the disease. Although it may not influence the course, in some cases, it appears to reduce the carrier stage. When virulent organisms persist, removal of adenoids and tonsils is advised.

MONTHLY MORBIDITY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	Sept. 1951	Sept. 1950	Jan.-Sept. 1951	Jan.-Sept. 1950
Brucellosis	12	8	66	47
Diarrhea and Dysentery	537	539	2,302	3,247
Diphtheria	12	30	88	132
Measles	65	64	13,746	2,540
Meningitis (Meningococcal)	8	1	95	92
Poliomyelitis	97	337	191	887
Rocky Mountain Spotted Fever	6	6	58	64
Scarlet Fever	39	55	742	644
Tularemia	3	8	33	33
Typhoid and Paratyphoid	7	11	41	68

BOOK ANNOUNCEMENTS

Spatial Vector Electrocardiography. By ROBERT P. GRANT, M.D., and E. HARVEY ESTES, JR., M.D., 1951. The Blakiston Company, Philadelphia, New York and Toronto. 41 Figures; 149 pages. Cloth. Price $4.50.

Manual of Tumor Nomenclature and Coding. Prepared by Subcommittee of the Statistics Committee of the American Cancer Society. 1951. 119 pages. Cloth. Price $2.50.

Master Your Mind. By SAMUEL KAHN, M.D., Ph.D. Rockport Press, Inc., New York. 1951. xxviii-262 pages. Cloth.

Let's Have Healthy Children. By ADELLE DAVIS, A.B., M.S., Consulting Nutritionist. Harcourt, Brace and Company, New York. 1951. 314 pages. Cloth. Price $3.00.

Let's Cook It Right. By ADELLE DAVIS, A.B., M.S., Consulting Nutritionist. Harcourt, Brace and Company, New York. 1951. x-626 pages. Cloth. Price $3.00.

The Changing Years. What to Do About the Menopause. By MADELINE GRAY. Doubleday & Company, Inc., Garden City, N. Y. 1951. 224 pages. Cloth. Price $2.75.

Technical Methods for the Technician. By ANSON LEE BROWN, B.A., M.D., Columbus, Ohio. Published by Anson L. Brown, Inc., Columbus, Ohio. 1950-51. xvi-784 pages. Illustrated. Cloth. Price $10.00.

Review of Physiological Chemistry. By HAROLD A. HARPER, PhD., Professor of Biology (Biochemistry), University of San Francisco; Lecturer in Surgery, University of California School of Medicine; etc. Third Edition. University Medical Publishers, Palo Alto, Calif. 1951. 260 pages. Price $3.50.

Patterns of Disease. On a Basis of Physiologic Pathology. By FRANK L. APPERLY, M.A., M.D. (Oxford), D. Sc. (Melbourne), F.R. C.P. (London), Professor of Pathology, Medical College of Virginia, Richmond, Virginia. J. B. Lippincott Company, Philadelphia. 1951. xiii-456 pages. 50 figures and 37 charts. Price $8.00.

This little volume, based on lectures on pathology delivered at the Medical College of Virginia, should prove valuable to those of us who have been out of school for some years and need bringing up to date on the newer ideas about disease, especially the virus and Rickettsial diseases and the part that chemistry plays in the formation of antibodies and in the processes of repair. Dr. Apperly is an excellent teacher and has a gift of explaining complicated processes. Take for instance his description of a virus. "It has been suggested that somewhere in past ages certain bacteria, living a sheltered parasitic existence in certain animals for many generations and bathed in a medium of predigested nutriment, gradually lost their own powers of digestion, i.e., their own enzymes necessary for independent existence and growth. This increasing shedding of its functions and consequent atrophy have finally reduced the parasite to such ultramicroscopic dimensions that in some cases it is doubtful if the parasite is even a living organism. At any rate, it is able to penetrate the cell, to fatten on its host, to be protected by it from outside antibodies and chemicals and thus to interfere in its host's metabolism—the complete parasite! This organism, if indeed we can so honor it, is called a *virus*." His description of phagocytosis is equally interesting. The leucocyte, driven by positive chemotaxis, approaches the bacterium and proceeds to feel, fondle and finally phagocytose its prey.

M.P.R.

Metabolic Methods. Clinical Procedures in the Study of Metabolic Functions. By C. FRANK CONSOLAZIO, Chief of Biochemistry, United States Army Medical Nutrition Laboratory, Chicago, Ill.; ROBERT E. JOHNSON, M.D., D. PHIL. (OXFORD), Professor and Head of the Department of Physiology, University of Illinois; and EVELYN MAREK, M.A., Biochemist, United States Army Medical Nutrition Laboratory. St. Louis, The C. V. Mosby Company. 1951. 470 pages. Illustrated. Cloth. Price $6.75.

A concise and accurate reference book of general and special procedures necessary for the conduct of metabolic studies and clinical investigation. Not only giving the explicit procedures for a multitude of chemical and physicial measurements on blood, serum, urine, and feces, the easy reading of physical principles involved in most of the complex instruments used in special and general clinical laboratories will greatly aid the chemist or technical assistant using this manual to understand instrumentation. Metabolic laboratories and clinical investigators actively using a great number of chemical determinations in the study of normal physiology or disease process will find this compilation into one volume a great time saver in methodology. The additional references given for the various procedures make it even more encompassing of metabolic methods. This excellent book, greatly needed for a long time, will quickly find many enthusiasts among clinical investigators and biochemists.

G.W.J. III

XUM

EDITORIALS

John Trible Thomas Hundley

OUR new president was born in Dunnsville, Essex County, Virginia on August 29, 1898. His father was John Trible Thomas Hundley, a minister of the Disciples of Christ Church, and for over 20 years the eminent president of Lynchburg College. His mother was Sue Fleet Walker.

Dr. Hundley received his A.B. degree from Lynchburg College in 1919 and was graduated as a doctor of medicine by the University of Maryland in 1923. Following his internship he practiced for a time in the coal fields at Lillybrook, West Virginia. In 1931 he returned to Lynchburg and has since confined his work to internal medicine in that city.

JOHN TRIBLE THOMAS HUNDLEY, M.D.,
President, The Medical Society of Virginia.

In 1924 Dr. Hundley was married to Katherine Elizabeth Swan of Baltimore. They have two children, Mrs. James Willard Greer of South Boston and John T. T. Hundley, Jr., who is married and resides in Lynchburg. There are three grandchildren.

Dr. Hundley has long been affiliated with civic endeavors in Lynchburg. Among his interests are the Kiwanis Club and the local Boy Scout Council. He served as chairman of the committee which organized the Lynchburg Guidance Center, later as its first president; also he acted in somewhat similar capacities for the Piedmont Hospital Service Association, as organizing chairman and as a member of its board.

He has been active in local, state and national medical affairs for many years. Thus, he has officiated in the following capacities: Secretary and President of the Lynchburg

Academy; Councilor for the Sixth District; as Vice-president of the Medical Society of Virginia; member of its Council executive committee; state chairman of the AMA Educational Campaign; and member and chairman of the Committee on Medical Economics.

While so generously serving organized medicine and participating in civic enterprises, Dr. Hundley has conducted an extensive medical practice. He has served on the staffs of the Lynchburg General Hospital, the Virginia Baptist Hospital, and the Memorial Hospital either as secretary or as chief of staff.

His principal interest in organized medicine during the past several years has been in the field of public relations. He has made valuable contributions to this work of the Society and some of his findings have been used nationally as a basis for improvement in medical service.

His efforts in the solution of socio-economic problems of medicine have also received recognition in national lay publications. He has often participated in non-medical discussion groups, ably representing the side of medicine.

Dr. Hundley is an energetic and enthusiastic worker for the betterment of medical care and medical service through improvement of the private practice of medicine. His findings, based upon detailed study, mature reflection, and consideration of the viewpoint of others, have been presented in a clear, forceful and convincing manner. From the platform, he speaks ably and with unusual clarity and conviction.

It is a significant fact that those who have known Dr. Hundley and his work the best have selected him for the Presidency of The Medical Society of Virginia by unanimous vote.

J.P.K.

The Diabetes Detection Drive

IT IS estimated that one million cases of diabetes are unrecognized in this country. The diagnosis of this disease rests upon a simple, quick laboratory test of the urine or the blood. It is so simple, in fact, that the patient himself can perform it and the results are unequivocal. Furthermore, the treatment of the disease is so satisfactory that there can be no objection to the drive, that the American Diabetes Association is conducting, on the ground of stimulating mass hysteria. Case finding campaigns in connection with many chronic diseases may, and indeed actually do, produce harm because of (1) uncertainty of the diagnosis and (2) the difficult and not-too-satisfactory treatment one has to offer the patient. This is not true in the case of diabetes. The recognition and treatment of diabetes is followed by improved health, and failure of recognition indeed may be disastrous. In case of accident or operation, an unrecognized diabetic runs a much greater risk than does one whose disease is being treated.

The diabetes detection drive has the approval of the American Medical Association. Every physician is requested to test his own urine and that of each member of his family today, and that of every patient routinely; second, to offer his assistance in the drive; third, to urge his Medical Society officers to form a Committee on Diabetes and recommend that every person in the community be tested. The principle of self-testing for glycosuria was approved by the Council of the American Diabetes Association, June 5, 1949, and by the House of Delegates of the American Medical Association, June 28, 1950. Self-testing units contain directions for use and instruction to report all positive findings to family physicians for confirmatory diagnosis.

XUM

What's Wrong With Modern Medical Education?

GENERAL Paul R. Hawley in the Irving S. Cutter Medalist Address (*Journal of Phi Rho Sigma,* **46**: Oct. 1950) made the startling, but nevertheless true, statement, "Never has the medical graduate been so poorly prepared as he is today—actually so completely unprepared—to face the complex problems with which he will be confronted the moment he enters private practice. It is a tragic paradox, that with a professional armamentarium never before equalled, today's medical graduate is handicapped in applying it to the general good of humanity by his ignorance of the social and economic structure within which he must serve." Never has the doctor's public relations been so poor. The public is now "Mad at Doctors", not one doctor, or certain doctors, but all doctors. This deterioration in public relation has taken place in the past forty years and General Hawley attempts to account for it.

It is due, he believes, to the changed attitude of the young doctor toward the patient. Forty years ago the welfare of the patient came first by long odds. Many a vacation was abandoned or postponed because some patient was too ill to leave. Whether the patient paid or not was distinctly a secondary matter. The patient's need was the main consideration.

The picture of the interne of forty years ago as drawn by General Hawley and the interne of today presents an even more startling contrast. Formerly, the pay consisted of room, board, and laundry, and the interne was content provided the training was good. Recently, this question was aired in the correspondence columns of the daily newspapers and not a word was said about the character of the training received. The discussion was confined to remuneration and "hours". Formerly, the interne took a night off every now and then if his work were up and he got another interne to take his calls. Now, General Hawley continues, the interne confers a favor on the hospital by accepting a living wage for a 40-hour week and nothing interferes with his hours off. Seemingly he has no feeling of responsibility for his patients. General Hawley also calls attention to another evidence of the young doctor's indifference to the patient's welfare and that is the patient's expenses. When unnecessary laboratory procedures are ordered and when the patient is allowed to stay in the hospital longer than necessary, it adds to the expense. It may be that the patient has prepaid hospital insurance, but this unnecessary expense will have to be paid by someone and will eventually be reflected in the rates some future patient will have to pay.

What has the full-time teacher to do with this problem? It seems reasonable that he has a great deal. To a private practitioner a patient is a person and is usually treated as such. To a full-time teacher a patient is a problem and is treated as such. Which attitude the new graduate takes depends upon the graduate and the hospital in which he internes. General Hawley suggests that the fraternities have a roll in determining this attitude. By informal discussion and smokers they can bring the undergraduate in contact with alumni who have successfully solved such problems. In such contacts the medical student may learn that there is a humanistic side to medicine which is as important as the scientific side.

Our State Board of Medical Examiners

THE function of the State Board is the protection of the public against persons who have not had adequate exposure to suitable training in the healing arts. At present the Board is composed of fourteen members: one doctor of medicine from each

congressional district, an homeopath, an osteopath, two chiropractors, and a naturopath. Recently a chiropodist has been added but as yet he has not functioned. Admittedly the law under which the Board works is illogical. It is the result of political compromise, but with it the Board has been getting satisfactory results.

Standing as it does, guarding the entrance to the practice of medicine, the Board is of concern to medical schools, the public, and the profession, and especially the new graduate. Before there were State Medical Examining Boards, medical education was in a chaotic condition. Medical schools varied from "diploma mills" all the way up the scale to first class institutions. The public had no way of knowing just what an M.D. degree meant. Sometimes it was merely a receipt for a fee paid and sometimes it represented hard and worthwhile work. Many of us can remember when the results of the Board's examinations were published in the newspapers with the number of applicants and failures per school. When this practice began, it was not long before the diploma mills closed up and the weaker schools improved and the results of the examinations ceased to be news. Medical education became more standardized. Practically everyone passed and the examination became, like the night stick of the London "bobby", just the symbol of authority.

The present law requires that an applicant for license to practice must be a graduate of a school at least the equal of the two medical schools in Virginia. Thus it has become the duty of the Board to examine the school as well as the graduate. In the case of refugee doctors this imposes considerable difficulties and may lead to court action as was recently the case in Southwest Virginia.

The public is inclined to look upon the Medical Practice Act and the examining Board as creatures of the medical profession. Nothing could be further from the truth. The members of the Board are appointed for five year terms by the Governor. In the case of the medical members, The Medical Society of Virginia submits to the Governor a list of three names from which he makes his appointment.

An important part of the duties of the Board is to see that the law is obeyed. It can revoke the license of anyone violating, for instance, the narcotic law or performing an illegal operation, and it can prosecute anyone practicing without a license.

The refugee doctor has given the Board a great deal of trouble. In the first place it is hard to evaluate his background and qualifications. Experience has shown that many are not trustworthy. Some have been admitted to practice in Virginia under pressure of some rural communities that needed doctors and invariably they have moved on to greener fields. This was the cause of breaking off reciprocity agreements with New York. Needless to say such varied duties demand sound judgment and ability.

The doctors of the Board are the first doctors in an official capacity that the new doctor meets on entering his life work. They are, in a way, a welcoming committee and they frequently make a lasting impression on the new comer. For this reason if for no other, they should be representative of our very best. Of such has the Board been composed in the past, and of such, we hope, that it will continue to be in the future.

The terms of two members, Dr. M. S. Fitchett of Norfolk and Dr. Guy Horsley of Richmond, expired this year. Dr. Horsley was the President of the Board. Both have served faithfully and well and the MONTHLY takes this occasion to thank them on behalf of The Medical Society of Virginia for this fine service they have rendered their State.

XUM

SOCIETY PROCEEDINGS

The Virginia State Board of Medical Examiners

Announces the following list of applicants who were licensed by the Board at its last regular meeting, June 13-16, 1951:

BY RECIPROCITY

Dr. Richard Jenks Ackart, Baltimore, Md.
Dr. Robert Harper Anderson, Alexandria.
Dr. William Cooke Andrews, Norfolk.
Dr. Thomas George Boisclair, Arlington.
Dr. George Joseph Carroll, Washington, D. C.
Dr. Edward Philip Cawley, Charlottesville.
Dr. Claude C. Caylor, Jr., Alexandria.
Dr. Conrad Clark Clement, Alexandria.
Dr. Charles Barrie Cook, Alexandria.
Dr. Bennett Y. Cowan, Philadelphia, Pa.
Dr. Jerome Edward Curtin, Arlington.
Dr. Cecil Burton Dixon, Paces.
Dr. Robert Edward duPrey, Washington, D. C.
Dr. Herman Lee Earnhardt, Jr., Radford.
Dr. William Clay Fritz, Franktown.
Dr. Clifford Garland Gaddy, Charlottesville.
Dr. Herbert Harold Galston, Richmond.
Dr. Milton Goldin, Portsmouth.
Dr. Henry Thomas Gray, Charlottesville.
Dr. Reginald Emerson Gregory, Altavista.
Dr. James Louis Guillette, Richmond.
Dr. Theron Henry Haas, Radford.
Dr. John Roland Hankins, Baltimore, Md.
Dr. John George Klemm Harvey, Danville.
Dr. Forrest Moseley Haswell, South Boston.
Dr. Richard L. Johnson, Roanoke.
Dr. James William Johnston, Richmond.
Dr. Paul Kaufman, Washington, D. C.
Dr. Robert Aloysius Kelley, Charlottesville.
Dr. Harold Nelson King, Hampton.
Dr. Ludwig C. Kroutil, Silver Spring, Md.
Dr. Daniel Leavitt, Roanoke.
Dr. Walter William Ledyard, Charlottesville.
Dr. Emmett Patrick Madigan, Bethesda, Md.
Dr. William Carey Meloy, Washington, D. C.
Dr. Arthur Albert Newbarr, North Garden.
Dr. James Arthur Newcome, Roanoke.
Dr. Charles Greylock Noble, Chincoteague.
Dr. Duncan Parham, Flushing, N. Y.
Dr. Julius Peerless, Norfolk.
Dr. Oliver James Purnell, Jr., Richmond.
Dr. John Lewis Read, Richmond.
Dr. Charles Henry Ross, Jonesville.
Dr. Paul Leslie Rowan, Richlands.
Dr. Soon Tai Ryang, Petersburg.
Dr. William A. Shafer, Bluefield, W. Va.
Dr. Peter Soyster, Falls Church.
Dr. Harrison Clark Spencer, Abingdon.
Dr. Andrew Tessitore, Washington, D. C.
Dr. John Wesley Todd, III, Rochester, Minn.
Dr. John Bacchus Truslow, Richmond.
Dr. John W. Uzmann, Roanoke.
Dr. Norman St. George Vann, Hampton.
Dr. Jack William Verran, Dunbar.
Dr. Charles Monroe Warr, Jr., Richmond.
Dr. Clayton Eugene Wheeler, Jr., Charlottesville.
Dr. Lester Arnauld Wilson, Jr., Charlottesville.
Dr. Leslie Norwood Winston, Richmond.
Dr. Jack Richard Woodside, Arlington.
Dr. Lewis H. Wright, New York, N. Y.

BY EXAMINATION

Dr. Albert Harold Arenowitz, Charlottesville.
Dr. Evan Hamilton Ashby, Jr., Charlottesville.
Dr. Ratcliffe Cox Barnes, Charlottesville.
Dr. Hubert Elmore Batten, Richmond.
Dr. Wayland Richard Bennett, Madison Heights.
Dr. Carlos Simon Berrocal, Puerto Rico.
Dr. William Jasper Berry, Oak Hill, W. Va.
Dr. Frank Nash Bilisoly, III, Charlottesville.
Dr. Ernst Moritz Blumenberg, Marion.
Dr. Lewis Booker, Union City, Ind.
Dr. Courtney Cox Bowen, Richmond.
Dr. Walter Morris Brady, Lynchburg.
Dr. Harvey Jerome Breit, Charlottesville.
Dr. Seymour Monty Brenner, Long Island, N. Y.
Dr. Joseph Herbert Britton, Richmond.
Dr. Raymond Keith Brown, Charlottesville.
Dr. John Chalkley Buchanan, Clintwood.
Dr. Ralph Burton Burner, Philippi, W. Va.
Dr. Ira Marshall Cantin, Virginia Beach.
Dr. Mariam Willena Carmichael, Richmond.
Dr. Floyd W. Carneal, Jr., Hampton.
Dr. Wallace Alexander Coburn, Seattle, Wash.
Dr. William Agee Cook, Jr., Madison Heights.
Dr. Philip Winston Coherd, Jr., Winchester.
Dr. Martel Jennings Dailey, Tye River.
Dr. Oscar Bruton Darden, Jr., Richmond.
Dr. Merle Burton Davis, Fairmont, W. Va.
Dr. Preston Caldwell Davis, Charleston, W. Va.
Dr. William Templeton Davis, Wilmington, N. C.
Dr. Paul Sahak Derian, New York City, N. Y.
Dr. Julia Cecil Dickinson, Richmond.
Dr. William S. Dingledine, Emory University, Ga.
Dr. Charles Coleman Donegan, Jr., Charlottesville.
Dr. Leonard Ned Ebin, Charlottesville.
Dr. Fred Ray Edens, Norton.
Dr. Adolphe Max Ehrenworth, Norfolk.
Dr. Warren Dunlap Elliott, University.
Dr. John L. Fairly, Jr., Richmond.
Dr. Cyrus Creston Farrow, Jr., Richmond.
Dr. Frederick A. Feddeman, Lexington.
Dr. Donald F. Fletcher, Horsey.
Dr. Clifford Henry Fox, Charlottesville.
Dr. Calvin Hunter Frazier, Miami, Fla.

Dr. Emma Jane Freeman, Adrian, W. Va.
Dr. William F. Gibbs, Norfolk.
Dr. Matthew C. Glynn, Portsmouth.
Dr. John B. Gorman, Lynchburg.
Dr. Richard Kelly Greenbank, Detroit, Mich.
Dr. William Hugh Grey, Richmond.
Dr. Anthony James Grieco, Charlottesville.
Dr. Francis Gordon Griffin, Windsor.
Dr. William C. Grigsby, Jr., Bristol.
Dr. Luther J. Hamlett, Richmond.
Dr. Robert Harris, Staten Island, N. Y.
Dr. Lewis Bailey Hasty, Richmond.
Dr. Robert Traylor Helmen, South Bend, Ind.
Dr. Philip Kearney Hensel, Camp Hill, Pa.
Dr. Norman Nash Hill, Jr., Norfolk.
Dr. Durell Boyd Horsley, Schuyler.
Dr. Wm. John Hotchkiss, Winchester.
Dr. Aubrey Alphin Houser, Jr., Richmond.
Dr. Austin Taber Hyde, Jr., Charlottesville.
Dr. Lawrence Perry Hyde, Swickley, Pa.
Dr. Allan Hammett Jefferies, Gaffney, S. C.
Dr. Eileen Thorpe Jennings, Appomattox.
Dr. Thomas Henry Jennings, Appomattox.
Dr. Walter Stanley Jennings, Fentress.
Dr. Albert McCray Jones, Norfolk.
Dr. Edward Paul Jones, Richmond.
Dr. Herbert C. Jones, Jr., Petersburg.
Dr. Ernest Jackson Keffer, Jr., Roanoke.
Dr. Louis Henry Keffer, Jr., Portsmouth.
Dr. Harold Kennedy, Martinsville.
Dr. James Moultrie Lee, Charlottesville.
Dr. Edward Arthur Lewis, Aylett.
Dr. Joseph J. Lindley, Richmond.
Dr. Henry Reece Little, Jr., Altavista.
Dr. Joseph Humphries Magee, Charlottesville.
Dr. Virginus Archer Marks, Richmond.
Dr. Erma Jean Marra, Richmond.
Dr. Arthur Joseph Martin, Highland Springs.
Dr. Homer Delmar Martin, Richmond.
Dr. James Dunn Mason, Jr., Petersburg.
Dr. Joseph Emory Mathias, Richmond.
Dr. Thomas Day McCahill, Worchester, Mass.
Dr. Joseph Frederick McGuire, Grundy.
Dr. William Francis McGuire, Charlottesville.
Dr. Elmer Francis McHugh, Pittsburgh, Pa.
Dr. Francis Hunter McMullan, Richmond.
Dr. Arthur W. D. Mears, Belle Haven.
Dr. Charles Samuel Miller, Charlottesville.
Dr. Harold W. Miller, Jr., Richmond.
Dr. Andrew C. Mitchell, Charlottesville.
Dr. Brooke Miller Moffett, Washington.
Dr. Hugh Kenneth Moir, Marion.
Dr. Garnett B. Moneymaker, Clifton Forge.
Dr. Pamela Ross Moore, Lewisburg, W. Va.
Dr. Robert Catchings Moore, Jr., Roanoke.
Dr. William Edward Newby, Richmond.
Dr. Abraham Lowell Newman, Williamsburg.
Dr. James Lee Northington, LaCrosse.
Dr. Allen Campbell Oglesby, Richmond.
Dr. Levi Old, Jr., London Bridge.
Dr. Wilmer Grey Oliphant, Mt. Hope, W. Va.
Dr. Bernard Oseroff, Norfolk.
Dr. Robert Macon Phillips, Petersburg.
Dr. William Herman Pifer, Birmingham, Ala.
Dr. Mary Martin Powell, Baltimore, Md.
Dr. Luke Ralph Rader, Richmond.
Dr. Charles Lankford Randolph, Jr., Richmond.
Dr. Robert Albion Repass, Bristol.
Dr. Harry H. Robinson, Charlottesville.
Dr. Carl J. Roncaglione, Richmond.
Dr. Charles Henry Sackett, Lynchburg.
Dr. Roy Preston Sandidge, Jr., Charlottesville.
Dr. Bernard Manuel Savage, Richmond.
Dr. Joseph Lee Sedwitz, Charlottesville.
Dr. Lewis Willard Shankel, Ft. Bliss, Texas.
Dr. William Henry Sipe, Charlottesville.
Dr. Victor Skorapa, Jr., Richmond.
Dr. Russell Smith, Roanoke.
Dr. Philip George Spaeth, Philadelphia, Pa.
Dr. Charles G. Spivey, Jr., Columbia, S. C.
Dr. John Miles Stirewalt, New Market.
Dr. Edward Jordan Stoll, Richmond.
Dr. James Walter Stone, Lynchburg.
Dr. Frances Milam Stoneburner, Sutherlin.
Dr. William Harold Stout, Charlottesville.
Dr. Albert Isadore Strauss, Brooklyn, N. Y.
Dr. Henry Morse Tanner, Jr., Charlottesville.
Dr. Richard Norvell Taylor, University.
Dr. Philip Randolph Thomas, Richmond.
Dr. Foxhall Parker Thornton, Jr., Elkton.
Dr. Norman Rock Tingle, Richmond.
Dr. Paul Ernest Totten, Charleston, W. Va.
Dr. Joe Watson Trent, Ft. Blackmore.
Dr. Jesse Miller Tucker, Jr., Richmond.
Dr. William Taylor Tucker, Charlottesville.
Dr. Harold Thomas Turner, Richmond.
Dr. Harry Charles Walker, Jr., Richmond.
Dr. Moffett R. Walker, Jr., Charlottesville.
Dr. Albert J. Wassermann, Richmond.
Dr. Julian Weinstein, Richmond.
Dr. William P. Wharton, Charlottesville.
Dr. James David Wilson, Clifton Forge.
Dr. Edward Woodward, Jr., Staunton.
Dr. Thomas Mahlon Wright, New York, N. Y.
Dr. Charles Murray Wylie, Richlands.
Dr. Harry Robert Yates, Jr., Charlottesville.
Dr. Yale Howard Zimberg, Richmond.

Norfolk County Medical Society.

At the last meeting before adjournment for the summer, Dr. Walter P. Adams succeeded to the presidency. Other officers elected are: President-elect, Dr. George A. Duncan; vice-president, Dr. Mallory S. Andrews; secretary, Dr. K. K. Wallace; and treasurer, Dr. J. Franklin Waddill. All are of Norfolk and the two latter were re-elected.

XUM

Roanoke Academy of Medicine.

Officers for the Academy, who took office October 1 are: President, Dr. Ira H. Hurt; president-elect, Dr. J. E. Gardner; vice-president, Dr. C. D. Nofsinger; and secretary-treasurer, Dr. Philip C. Trout. All are of Roanoke. Dr. A. P. Jones was named chairman of the Executive Committee; Dr. D. S. Garner, chairman of the Judicial and Ethics Committee; and Dr. M. A. Johnson, Jr., chairman of Public Relations.

The Fairfax County Medical Society,

At is September meeting, elected the following officers for the ensuing year: President, Dr. W. L. Harris, Falls Church; vice-president, Dr. T. B. McCord, Fairfax; treasurer, Dr. E. Newman, Vienna; and secretary, Dr. C. E. Cooper (re-elected), Annandale.

The Warwick County Medical Society

Held its first fall meeting at The James River Country Club in September with Dr. J. W. Carney, of Newport News, vice-president, presiding. There were fifty-six members and eleven guests present.

Dr. John P. McGovern, Assistant Professor of Pediatrics at The George Washington University School of Medicine in Washington, D. C., was the guest speaker. His subject was "Fluid and Electrolyte Management in Infants and Children."

Dr. William H. Woodson and Dr. O. Watts Booth, both of Newport News, are president and secretary, respectively, of this Society.

Rockbridge County Medical Society.

At the meeting of this Society on September 12, the following officers were elected: President, Dr. Hunter McClung, Lexington; vice-president, Dr. T. F. Kennan, Raphine; and secretary, Dr. Lewis A. Micou, Buena Vista. At this meeting two doctors were accepted as new members.

The Lynchburg Academy of Medicine

Held its regular meeting on September 10 at the Virginian Hotel, following dinner. Several new members were received at this time. The speaker of the evening was Dr. Kenneth Crispell, associate professor of Medicine at the University of Virginia, who spoke on Thyrotoxicosis.

Dr. J. W. Davis, Jr., is president, and Dr. Edwin A. Harper, secretary. Both are of Lynchburg.

NEWS

Executive Secretary-Treasurer and Director of Public Relations, Medical Society of Virginia.

The Committee appointed to Select a Successor to Mr. Henry S. Johnson, interviewed numerous candidates for this position, and after careful consideration decided on Mr. Robert I. Howard, provided he could be released from the Armed Forces.

Mr. Howard was Mr. Johnson's assistant prior to recall into the Army Air Force, and is therefore thoroughly familiar with office routine and problems peculiar to this type of medical organization.

The committee immediately started to work on Mr. Howard's release with the result that he assumed office October 5, 1951. We feel fortunate in having him with us again to head up the headquarters activities, since he is so eminently qualified, both as to personal attributes and experience.

J. M. EMMETT
GUY W. HORSLEY
JOHN T. T. HUNDLEY
VINCENT W. ARCHER, *Chairman*

The Medical Society of Virginia.

The 104th annual meeting of the Society, held at The Cavalier at Virginia Beach, October 7-10, under the presidency of Dr. C. Lydon Harrell of Norfolk, was a credit to the local committee and most successful in every way. There was a registered attendance of 638 doctors, 177 ladies, and 116 exhibitors. A number of affiliated societies held meetings at this time. Many matters of current interest were discussed.

The guests included Dr. Winthrop M. Phelps of Baltimore who spoke on General Management of the Cerebral Palsy Problem; Dr. Louis H. Bauer, President-Elect of the American Medical Association, who participated in the panel discussion "You and Your A.M.A."; Honorable Hugh D. Scott, a Virginian who went many years ago to Pennsylvania and is now a member of the U. S. House of Representatives. Mr. Scott spoke of a recent visit to the Western German zone, seeing the Russians having a peace celebration, of the interest of many in the United States, and also of a visit to General Eisen-

hower. The other guests were Dr. Theodore Woodward, Baltimore, who talked on Bacterial Diseases; and Dr. John E. Howard, also of Baltimore, whose subject was the Diagnosis and Treatment of Various Types of Spontaneous Hypoglycemia.

The exhibits, both technical and scientific, were excellent. In the scientific group, awards were given Dr. T. Addison Morgan of Franklin, Dr. William Parson, Dr. K. R. Crispell and Group of Charlottesville: and Dr. Robert L. Payne, Jr., Norfolk.

The Society voted to nominate to the American Medical Association at its December meeting Dr. W. C. Caudill of Pearisburg as their choice for the Physician of the year.

Dr. John T. T. Hundley of Lynchburg succeeded to the presidency and the following officers were named: President-elect, Dr. James L. Hamner, Mannboro; vice-president, Dr. Ira L. Hancock, Creeds, Dr. Walter P. Adams, Norfolk, and Dr. Mary Elizabeth Johnston, Tazewell; executive secretary-treasurer, Robert I. Howard, Richmond.

Councilors were named for the odd numbered districts, in accordance with the By-Laws, those for the even numbered districts holding over. Those named were Dr. A. A. Creecy, Newport News; Dr. Guy W. Horsley, Richmond; Dr. L. P. Bailey, Nathalie; Dr. H. W. Miller, Woodstock; and Dr. James P. King, Radford.

Dr. J. M. Hutcheson and Dr. Carrington Williams of Richmond hold over as delegate and alternate to the A.M.A. The new delegates are Dr. Vincent W. Archer, Charlottesville, and Dr. M. H. Harris, West Point. Their alternates are Dr. Kinloch Nelson, Richmond, and Dr. John O. Boyd, Jr., Roanoke.

The Society voted to hold the 1952 meeting in Richmond.

News from State Department of Health.

Health Services have been established in Lee and Scott Counties forming the Lee-Scott Health District with Dr. W. A. Werner as Health Officer. Headquarters for this district are in Gate City with a branch office in Jonesville.

Dr. Robert W. Jessee, former Health Officer of the Carroll-Grayson Health District and Dr. Charles M. Wylie, former Health Officer of the Tazewell-Buchanan District, have received scholarships to Johns Hopkins University where they will work on their Masters degrees in Public Health. They will return to the State Health Department in June of 1952.

Dr. A. R. Wilson Climie has been appointed Health Officer of the Alleghany-Botetourt Health District with headquarters in Covington.

Dr. William V. Harrison has been assigned as Health Officer to the Carroll-Grayson Health District with headquarters at Galax.

Dr. Joseph M. Rose has assumed duties as Health Officer of the Pittsylvania Health District with headquarters in Chatham.

Dr. R. B. Blackwell resigned as Health Officer of Buena Vista-Lexington-Rockbridge Health District effective October 15. Dr. Blackwell has accepted the position as Health Officer of the city of Newport News.

News From University of Virginia, Department of Medicine.

"Obstetrics and Gynecology in General Practice" was the subject of a conference at the University of Virginia Department of Medicine, October 19-20. This symposium was the first of a series during the academic year that is designed especially for general physicians, and planned also for those who wish credit toward post-graduate training for membership in the Virginia Academy of General Practitioners.

A conference on "The Management of Common Traumatic Injuries" will be held at the University on January 11, 1952, and a conference on "Infectious Diseases", April 4, 1952.

The School of Clinical Pathology of the University of Virginia Department of Medicine will affiliate with Mary Washington College of the University of Virginia in a progam of training for medical technologists. Candidates for B.S. degree in medical technology will spend three years in academic study at Mary Washington College and one year at the University Hospital.

Dr. William E. Bray, Professor of Clinical Pathology, has emphasized that the training his school gives to students from other colleges and universities will continue. These students, he said, are admitted to the course upon completion of two years college work and are awarded a certificate from the American Society of Clinical Pathologists when they have met all requirements of the society and passed examinations, but are not eligible for the degree.

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Dr. Walter O. Klingman has been promoted from Associate Professor to Professor of Neurology and Psychiatry. Other promotions recently announced are those of Dr. Kenneth R. Crispell to Assistant Professor of Internal Medicine, Dr. Robert A. Kelley to Assistant Professor of Urology, Dr. Preston B. Lowrance to Assistant Professor of Internal Medicine, Dr. Hans Hoch to Assistant Professor of Biochemistry, Dr. Clifford G. Gaddy to Instructor in Internal Medicine, and Dr. Donald Ferguson to Instructor in Internal Medicine.

Announcement has been made also of the appointment of Dr. William H. Wood, Jr., and Dr. Armistead Page Booker as Clinical Instructors in Pediatrics.

Dr. Myron M. Nichols, former Instructor in the Department of Pediatrics of the University of Arkansas and Pediatric Consultant to the Arkansas State Board of Health, began serving the first of July as Instructor in Pediatrics, University of Virginia Department of Medicine.

Dr. William Edmund Craddock, Radiological Registrar of the X-Ray Department, University College Hospital, London, England, became associated with the University as Instructor in Roentgenology the first of July.

Major Roger J. Reynolds, former Chief of the Medical Service of the Army Hospital, Ft. McPherson, Ga., will direct the Medical R.O.T.C. of the University of Virginia while serving as Assistant Resident in Internal Medicine at the University Hospital.

Commemorating the 100th Anniversary of the birth of Walter Reed, University Department of Medicine graduate in 1869, and opening the Friday Evening Medical Lecture Series, Dr. William B. Bean, Professor of Internal Medicine of the University of Iowa, spoke October 5, on "Walter Reed, the Study of a Disciplined Mind".

The second of twenty-one speakers of the Friday Evening Medical Lecture Series was Dr. George A. Perera, Department of Medicine, Columbia University, who spoke October 12, on "Essential Hypertension and Its Endocrine Relationships".

The third lecturer, Dr. F. Bayard Carter, Department of Obstetrics and Gynecology, Duke University, spoke October 19, on "The Menopause". This was the Tiffany J. Williams Memorial Lecture given in Conjunction with the Post Graduate Conference on Obstetrics and Gynecology in General Practice.

Dr. Julius H. Comroe, Jr., Department of Physiology and Pharmacology University of Pennsylvania, spoke October 26, on "Dyspnea".

Other lectures scheduled for the fall program include those by Dr. Alan Moritz, Institute of Pathology, Western Reserve University, on "The Medical and Medico-Legal Implications of Unexplained Deaths", November 2; Dr. Richard J. Bing, Johns Hopkins University, on "Some New Aspects of the Physiology of the Human Heart", November 9; Dr. Clarence C. Little, Jackson Memorial Laboratory, Bar Harbor, Maine, on "Genetics in Relation to Basic and Clinical Research in Cancer", November 30; Dr. Alton Ochsner, Tulane University School of Medicine, on "The Early Diagnosis and Treatment of Gastric Carcinoma", December 7.

A federal grant of $485,000, from Hill-Burton Funds has been awarded the University Hospital for expansion in the hospital and erection of new kitchen facilities.

Dr. Osborne Honored.

Dr. Archibald P. Osborne of Berryville was honored by the citizens of Clarke County on October 1, when they tendered him and Mrs. Osborne a reception in the parish house of the Episcopal church. This marked the beginning of his fiftieth year of medical practice in that county, and was also the golden wedding anniversary of the couple.

Dr. Joseph F. Wilson,

Class of '44, Medical College of Virginia, after service of more than a year in Korea, joined the staff of McGuire Hospital, Richmond, on October 1, as an assistant in surgery. He is a son of Dr. Robert M. Wilson who directed a leper colony in Kwangju, Korea, from 1908 to 1941, and later was Henrico County health officer. Dr. Joseph Wilson was born in Kwangju.

New Health Officer at V.P.I.

Dr. Charles Roger Lyons has been appointed health officer at Virginia Polytechnic Institute, effective October 1. He replaces Dr. Charles R. Woolwine who retired August 31 after serving for twenty-three years. Dr. Lyons comes from Montana State University, where he had been health officer since 1947.

Richmond Board of Health.

Dr. M. Pierce Rucker will serve as chairman of the Board for the coming year, and Dr. Emily Gardner will be vice-chairman.

Dr. Harry J. Warthen has been named for a new term on the Board.

Medical College of Virginia News.

On September 26, a portrait was presented to the College of Dr. C. C. Coleman, who recently resigned as head of the department of neurological surgery. The portrait was presented by Dr. Frank H. Mayfield of Cincinnati, who trained under Dr. Coleman at the College, and it was received on behalf of the College by Dr. W. T. Sanger, president. Tributes were paid by Dr. I. A. Bigger, Dr. J. M. Hutcheson and Dr. John Bell Williams. The portrait was unveiled by Dr. Coleman's daughters. The ceremonies were attended by several hundred friends and medical associates of Dr. Coleman.

Dr. John M. Meredith has been named as professor and head of the department of Neurological Surgery at the College, succeeding Dr. Coleman, resigned. Dr. Meredith is an alumnus of the University of Pennsylvania, class of '30, and was on the teaching staff of the University of Virginia before becoming associated with Dr. Coleman in 1941.

Ground was broken for the new building for clinical dentistry at the Medical College of Virginia on September 11. The first two floors of the building are expected to be completed within the next 12 to 15 months and will provide space for expanded and newly equipped clinic facilities. The remaining two floors to house technic and research laboratories, lecture rooms and demonstration clinics are awaiting construction pending appropriations from the General Assembly.

The Thermal and Radiation Panel of the Research and Development Board, Department of Defense, met at the Medical College of Virginia on September 18. This group of civilian and military personnel under the chairmanship of Dr. Joseph Aub, professor of medicine at Harvard University, studied the present results of the work on burn research directed by Dr. Everett I. Evans and considered future plans.

Dr. Evans delivered the 26th Da Costa oration at the Philadelphia Academy of Medicine on September 26 on "The Management of Severe Burns".

Dr. William B. Porter, professor of medicine, delivered five lectures before the Medical Association of Puerto Rico in San Juan on heart disease. He also held four clinical conferences at the Municipal, Presbyterian, and Veterans Administration Hospitals there between September 1-12.

Dr. William T. Sanger, president of the Medical College of Virginia, was made president-elect of the National Society for Crippled Children and Adults at the annual convention in Chicago October 3-5.

Dr. Ralph M. Tovell, chief of the Department of Anesthesiology of Hartford Hospital, spoke to members of the staff on October 3 on the subject, "Local Anesthesia".

The enrollment for the session 1951-52 in the School of Medicine is 365; in the School of Dentistry, 202; in the School of Pharmacy, 218; in the School of Nursing, 180. There are 46 students in Physical Therapy; 8 in Hospital Administration; 24 in X-ray Technology; 15 in Medical Technology; and 3 in Public Health Nursing. In addition there are 119 interns and residents, 12 dietetic interns, 20 graduate students, and 57 nurse affiliates. Students enrolled in the Saint Philip unit for Negro women included 91 in the basic nursing course, 5 in Public Health Nursing, and 62 affiliates.

Dr. Richard Fowlkes,

Professor of Dermatology, Medical College of Virginia, Richmond, was elected President of the South-Eastern Dermatological Association at its meeting in Birmingham, Alabama, on September 2. The next meeting of the organization will be held in Nashville, Tennessee.

Dr. Arthur H. Ruggles to Give Salmon Lectures.

The Salmon Committee on Psychiatry and Mental Hygiene announces that Dr. Arthur Hiler Ruggles will deliver this year's Thomas William Salmon Lectures at the New York Academy of Medicine. His subject will be "The Place and Scope of Psychotherapy", and the talks will be given on the evenings of November 7, 14 and 20 at 8:30 o'clock. Dr. Ruggles is a former President of the American Psychiatric Association and of the National Committee for Mental Health. During World War II he was Consultant to the Secretary of War and Chairman of the Committee on War Psychiatry. He is at pres-

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ent General Consultant to Butler Hospital, Providence, R.I., of which he was Superintendent for 26 years, and Special Consultant to the Emma Pendleton Bradley Home in Providence.

New State Medical Journal.

The Medical and Chirurgical Society of Maryland will publish a monthly state medical journal beginning January 1952. It will be known as the *Maryland Medical Journal* and will serve as the official organ of that state society.

Gill Memorial Eye, Ear and Throat Hospital Residents.

The annual meeting of the former residents of the Gill Memorial Eye, Ear and Throat Hospital, Roanoke, was held at the hospital on September 17, at which time the following papers were presented:

"Foreign Bodies of the Maxillary Sinus"—Dr. Paul T. Meyers, Johnstown, Pa.

"Cancer of the Cornea with Multiple Skin Cancers"—Dr. G. A. Smith, Montgomery, W. Va.

"Melanosarcoma of Choroid"—Dr. R. M. Ferrell, Lewisburg, W. Va.

"Surgical Management of Crossed Eyes"—Dr. E. G. Gill, Roanoke.

"Rhinoplastic Correction of the Enlarged and the Depressed Nose"—Dr. Frederick D. White, Roanoke.

"Technique and End Results of Operation for Deafness"—Dr. Houston L. Bell, Roanoke.

The officers of the association are: President, Dr. F. Buerk Zimmerman, Louisville, Kentucky; Vice-Presidents, Dr. Keith Gerchow, Morgantown, W. Va., and Dr. R. M. Ferrell, Lewisburg, W. Va.; Secretary-Treasurer, Dr. H. L. Bell, Roanoke.

The Stuart McGuire Lecture Series

And Symposium on Cardiac Surgery and Cardiology will be held November 7-9, in the Egyptian Building of the Medical College of Virginia, Richmond. There is no charge for the McGuire Lectures, but a charge of $5 a day for the lectures to be given on the 8th and 9th, except to members of the faculty of the Medical College of Virginia, the Medical Department of the University of Virginia, physicians of McGuire Veterans Hospital, medical students, and members of the house staff of any hospital. Visiting lecturer will include Drs. Charles P. Bailey, John H. Gibbon, Jr., and Redondo-Ramirez of Philadelphia; Dr. George E. Burch, New Orleans; Dr. Daniel F. Downing, Newark, N. J.; Drs. Richard W. Eckstein and Harold Feil of Cleveland; Dr. Donald E. Gregg, Washington; and Dr. Irving S. Wright, New York.

The Post Graduate Clinical Sessions,

Sponsored by The Virginia Society of Ophthalmology and Otolaryngology, will be held in Richmond, at the Baruch Auditorium of the Medical College of Virginia, November 27, 28, 29 and 30, 1951. The first two days are for otolaryngology and the remaining two days for ophthalmology.

For further information inquiries may be addressed to P. N. Pastore, M.D., Chairman, Box 25, Medical College of Virginia Station, 1200 East Broad Street, Richmond 19.

Dr. William B. McIlwaine,

Petersburg, went to Boston about the first of October to take a graduate course at Harvard Medical School in pediatric endocrinology.

Two New Hospitals in Virginia.

The Louise Obici Hospital was dedicated at Suffolk on September 16. This is a 100-bed hospital as a memorial to Mrs. Louise Obici, wife of the founder of the Planters Nut and Chocolate Company of that city. There will also be an adjoining building for the nurses and the class rooms and laboratories for the school for nurses.

The Shenandoah County Memorial Hospital was also dedicated on September the 16th. This is located at Woodstock and cost $800,000.

Urology Award.

The American Urological Association offers an annual award of $1000.00 (first prize of $500.00, second prize $300.00 and third prize $200.00) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to men in training to become urologists.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Chalfonte-Haddon Hall, Atlantic City, June 23-26, 1952.

For full particulars write the Secretary, Dr. Charles H. de T. Shivers, Boardwalk National Arcade Building, Atlantic City, New Jersey. Essays must be in his hands before February 15, 1952.

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Motion Pictures on Health.

A revised list of "Sources of Motion Pictures on Health" has been prepared by the Committee on Medical Motion Pictures of the American Medical Association. This new mimeographed list includes 9 pages of addresses of the major loan and rental libraries, the state health departments' film libraries and references to printed lists and catalogs. Copies are available from: Committee on Medical Motion Pictures, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

Van Meter Prize Award.

The American Goiter Association again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association which will be held in Saint Louis, Missouri, May 1, 2 and 3, 1952, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten double spaced copy in duplicate sent to the Corresponding Secretary, Dr. George C. Shivers, 100 East Saint Vrain Street, Colorado Springs, Colorado, not later than March 1, 1952.

The American Laryngological, Rhinological and Otological Society,

Southern Section, will meet in Atlanta at the Academy of Medicine, Monday, January 14, 1952. This will be a one-day meeting and there will be six speakers, each of whom is preeminent in his own field. All members of the Medical Profession are cordially invited. There is *no* registration fee. The vice-president in charge of the Southern Section is Dr. Lester A. Brown of Atlanta, Georgia.

Auto Accident Information Wanted—

If the doctor who accidentally backed his auto into side of my black Olds. 88 Sedan, on morning of October 10th, in near front of Cavalier Hotel, at Virginia Beach meeting, will kindly advise me his Insurance Carrier, same will be appreciated. Address Dr. W. B. Barton, Stonega, Virginia. (*Adv.*)

OBITUARIES

Dr. William Thomas McLemore,

For more than fifty years a practicing physician at Courtland, died September 29, at his home there. He was a life long resident of Southampton County and was graduated in 1899 from the former University College of Medicine, Richmond. He was a member of the Medical Society of Virginia in which he belonged to the "Fifty Year" club. His wife and a son survive him.

Dr. John Garnett Broaddus,

Prominent physician of Bowling Green, died September 19, of a heart attack while at a Richmond theater. He was seventy years of age and a graduate of the former University College of Medicine, Richmond, in 1905. After graduate work in New York and a short time in West Virginia, he returned to Bowling Green to enter practice with his father and had continued there for forty years. He was a member of the Medical Society of Virginia. Surviving are his wife and two daughters.

Dr. Wycliffe Charles Jackson,

Who practiced for twenty years in Tazewell County, died at the home of his mother in Amelia County, September 17, aged forty-seven. He was graduated from the Medical College of Virginia in 1928, and was a member of the Medical Society of Virginia. Beside his mother, he is survived by his wife and three sons.

Dr. William Edgar Waddell,

A prominent surgeon in Lexington, died October 7, after a long illness, aged 41. A native of that city, he graduated in medicine from the University of Virginia in 1934. After a short service in hospitals in Farmville and Norfolk, he joined the staff of Twillingate Hospital in Newfoundland as assistant surgeon. He served with the Army Medical Corps for nearly four years, during World War II, and was awarded both the Silver Star and Purple Heart. He returned to Lexington in 1946. He was a member of the Medical Society of Virginia and

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of the American College of Surgeons. His parents, his wife and two children survive him.

Dr. Theron Rice Rolston,

A physician at New Hope for twenty-two years, died September 23 at the age of forty-nine. He graduated from the Medical College of Virginia in 1929, was a former president of the Augusta County Medical Association, and was for some years a member of the Medical Society of Virginia. He was an elder in the Presbyterian Church and a member of the Augusta County School Board. Surviving are his wife and three children.

Dr. George McLean Lawson,

Professor of preventive medicine and public health at the University of Virginia, Charlottesville, died September 20, after an illness of two years. He was fifty-three years of age and a graduate in medicine from Yale University in 1924 and from Harvard in public health service in 1932. He joined the faculty at the University in 1937, was secretary of the medical faculty and was active in affairs of the medical school. He was a former member of the Medical Society of Virginia but resigned as he was not in practice. His wife and a son survive him.

Resolutions on Death of Henry S. Johnson.

The Richmond Public Relations Association of the Virginia State Chamber of Commerce, in session September 26, unanimously adopted the following:

BE IT RESOLVED that in the passing of Henry Satterwhite Johnson, a faithful member who worked enthusiastically and unceasingly in our Association, the Richmond Public Relations Association has sustained a profound loss;

BE IT RESOLVED that the members of this Association express their sincere sympathy to Mr. Johnson's family in their bereavement;

BE IT FURTHER RESOLVED that a copy of these resolutions be spread on the minutes of our Association, a copy sent to the VIRGINIA MEDICAL MONTHLY.

J. ROBERT ANDERSON
Chairman of Committee

Dr. Wilson Eliot Driver,

For many years a prominent eye, nose and throat specialist in Norfolk, died at a hospital in that city, October 14, after an illness of several months. He was eighty years of age and a graduate in medicine from the University of Maryland in 1893 and was one of the founders of the Norfolk General Hospital. Dr. Driver was a member of The Medical Society of Virginia for many years until his retirement from active practice in December 1941. He is survived by three children.

Dr. William Bradford Newcomb.

The Norfolk County Medical Society adopted the following in appreciation of the services of Dr. Newcomb:

Dr. William Bradford Newcomb, a prominent physician of Norfolk and a member and past president of this Society, died at his residence August 1, 1951.

A native of Gloucester County, Virginia, he received his academic education there. In 1906, he obtained a B.A. degree from the College of William and Mary. In 1910, he graduated from the Johns Hopkins School of Medicine. In 1913, after post-graduate study, he began the practice of Internal Medicine in Norfolk as an associate of Drs. Ruffin and Gwathmey. He maintained his office at Saint Christopher's Clinic until his recent illness. While confining his practice to Internal Medicine, Dr. Newcomb was well versed in all fields of the profession. In Pathology, he took special interest and was the guiding influence in the progress of the Pathological Department of the Norfolk General Hospital.

Dr. Newcomb was a member of The Medical Society of Virginia, the American Medical Association, the Seaboard Medical Society, and the American College of Physicians.

In World War I, Dr. Newcomb served on the Selective Service Advisory Board and, in World War II, organized a special reserve unit of the U. S. Public Health Service.

A lover of sports in general, Dr. Newcomb was an ardent horseman and through the organizations of Princess Anne Hunt Club and the Horse Show Associations, he did much to stimulate the interest in horsemanship in Virginia.

Dr. Newcomb was not married. He is survived by one sister, Mrs. Hunter Gates of Richmond and one brother, Dr. John Lloyd Newcomb, the retired President of the University of Virginia.

WHEREAS, we have been called upon to record the loss of Dr. Newcomb.

BE IT RESOLVED, that in his passing, our Society has lost a most esteemed member and the profession an outstanding counsellor.

BE IT RESOLVED, that we, the members of the Norfolk County Medical Society, extend to his family, our sympathy in their grief.

BE IT FURTHER RESOLVED, that a copy of these resolutions be spread upon the minutes of our Society as a permanent record, and that a copy be sent to the VIRGINIA MEDICAL MONTHLY and a copy to members of his family.

Committee:
G. R. BERKELEY, M.D.
A. B. HODGES, M.D.
W. A. PORTER, M.D., *Chairman*

An Advertisement of G. D. Searle & Co.

(The Council on Pharmacy and Chemistry of the American Medical Association has adopted the following statement of Actions and Uses and of Dosage for publication in connection with a description of Banthine Bromide for inclusion in New and Nonofficial Remedies)

METHANTHELINE BROMIDE.—*Banthine® Bromide (Searle)*

β-diethylmethylaminoethyl 9-xanthenecarboxylate bromide

Actions and Uses.—Methantheline bromide, a parasympatholytic agent, produces both the peripheral action of anticholinergic drugs such as atropine and the ganglionic blocking action of drugs such as tetraethylammonium chloride. Tolerated amounts of methantheline bromide exert side effects typical of atropine-like drugs, but cause less tachycardia, and also less postural hypotension than does tetraethylammonium chloride. Toxic doses produce a curare-like action at the somatic neuromuscular junction.

Clinical studies indicate that the drug effectively inhibits motility of the gastrointestinal and genitourinary tracts and, to a variable degree, diminishes the volume of perspiration and salivary, gastric and pancreatic secretions. It also decreases mucoprotein secretion. Like atropine, it produces mydriasis and cycloplegia when applied locally to the eye or administered systemically, but until more clinical evidence becomes available, its local use for this purpose is not recommended. The value of the drug for preventing abnormal cardiac reflexes through the vagus during thoracic surgery, or as an agent for routine preoperative medication in place of atropine, requires further investigation before final conclusions can be reached.

Methantheline bromide is indicated for clinical use whenever anticholinergic spasmolytic action is desired, provided it is not contraindicated because of its atropine-like characteristics or because of a patient's intolerance to the unavoidable side effects of such therapy. It is useful as an adjunct in the management of peptic ulcer, chronic hypertrophic gastritis, certain less specific forms of gastritis, pylorospasm, hyperemesis gravidarum, biliary dyskinesia, acute and chronic pancreatitis, hypermotility of the small intestine not associated with organic change, ileostomies, spastic colon (mucous colitis, irritable bowel), diverticulitis, ureteral and urinary bladder spasm, hyperhidrosis or control of normal sweating which aggravates certain dermatoses, and control of salivation.

Methantheline bromide produces some degree of cycloplegia and mydriasis in therapeutic doses and therefore should not be administered to patients with glaucoma. It sometimes decreases the ability to read fine print. Xerostomia (dryness of the mouth) is a common, sometimes transient, side effect. Urinary retention of varying degree may occur in elderly male patients with prostatic hypertrophy, and some patients may have difficulty emptying the rectum. Patients with edematous duodenal ulceration may experience nausea and vomiting during initial administration of the drug. These patients should take only liquids during the institution of drug therapy. All patients should be advised of the possible occurrence of side effects. Overdosage sufficient to produce a curare-like action may be counteracted by prompt subcutaneous injection of 2 mg. of neostigmine methylsulfate.

Dosage.—Methantheline bromide is administered orally or parenterally by either the intramuscular or intravenous route. Parenteral administration is not advised for patients able to take the drug orally. The average initial adult dose, oral or parenteral, is 50 mg. For patients with considerable intolerance, 25 mg. may be employed. In the management of peptic ulcer, a beginning schedule of 50 mg. three times daily before meals and 100 to 150 mg. on retiring is suggested. However, the usual effective dose is 100 mg. four times daily, although some patients may require more or less than this amount. The dosage may be increased to tolerance, using dryness of the mouth as a guide, and adjusted to meet the individual response of patients. Maintenance dosage in peptic ulcer is usually considered to be about one-half the therapeutic level. In the management of other hypermotile or hypersecretory states, the dosage should be adjusted to the smallest amount which will relieve the symptoms. When spastic conditions are secondary to inflammatory or other organic lesions, therapy directed toward the cause should be employed whenever possible.

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Orthopedic Surgery
WM. TATE GRAHAM, M.D.
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BEVERLEY B. CLARY, M.D.
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W. HUGHES EVANS, M.D.
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